SPECIAL PATHOLOGY AND THERAPEUTICS OF THE DISEASES OF DOMESTIC ANIMALS

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Table of Contents

Diseases of the Digestive Organs.

SECTION I.

Diseases of the Buccal Cavity.

PAGE

1. Catarrh of the Buccal Cavity. Stomatitis catarrhalis...... 1
2. Vesicular Inflammations of the Mouth. Stomatitis vesiculosa. 6
3. Aphthous Inflammation of the Mouth. Stomatitis aphthosa.. 9
   Aphthous Inflammation of the Mouth of the Young....... 9
   Infectious Papulous Inflammation of the Mouth of Cattle, Stomatitis papulosa bovum infectiosa.................. 12
   Other Diseases Similar to Stomatitis..................... 14
4. Thrush. Stomatitis oidica.............................. 17
5. Phlegmonous Inflammation of the Mouth. Stomatitis phlegmonosa 19
6. Ulcerative Inflammation of the Buccal Cavity. Stomatitis ulcerosa 21
   Other Diphtheroid Inflammations of the Mucosa of the Mouth 24
   Stomatitis gangraenosa................................. 25

SECTION II.

Diseases of the Salivary Glands.

1. Ptyalism 27
2. Parotitis 28
   Inflammation of the Submaxillary Gland................. 30
   Angina Ludovici.. 30

SECTION III.

1. Pharyngitis 31
2. Animal Parasites in the Pharynx.......................... 44
3. Tumors in the Throat. Tumores intra-et peripharyngeales... 45

SECTION IV.

Diseases of the Esophagus.

PAGE

1. Esophagitis 48
2. Spasm of the Esophagus. Esophagismus 50
3. Paralysis of the Esophagus. Paralysis oesophagi 52
4. Obstruction of the Esophagus. Obstructio oesophagi 53
5. Constriction of the Esophagus. Stenosis oesophagi 59
6. Dilatation of the Esophagus. Dilatatio oesophagi 63
7. *Neoplasms in the Esophagus. Tumores oesophagi* 68
8. Spiroptera sanguinolenta in the Esophagus 68
   Other Animal Parasites in the Esophagus 70
9. Diseases of the Crop in Fowl 70
   (a) Catarrh of the Crop 70
   (b) Occlusion of the Crop. Obstructio ingluviei 71
   (c) Animal Parasites in the Crop 73

SECTION V.

Diseases of the Stomach and Intestines.

1. Vomiting. Vomitus 74
2. Diseases of the Fore-stomachs (Rumen, Reticulum and Omasum) in Ruminants 79
   (a) Dilatation of the Rumen. Dilatatio acuta ruminis ab alimentis 80
   (b) Bloating of Ruminants, Acute 86
   (c) *Bloating of Ruminants, Chronic* 93
   (d) Atony of the Fore-stomachs. Atonia ruminis, reticuli et omasi 95
   Acetonemia in Cattle 105
   (e) Foreign Bodies in the Fore-stomachs. Corpora aliena reticuli, ruminis et omasi 108
   Other Inflammatory Diseases of the Fore-stomachs ... 123
3. Acute Catarrh of the Stomach. Catarrhus ventriculi acutus .. 124
4. Chronic Gastric Catarrh. Catarrhus ventriculi chronicus 132
   Gastritis Hyperplastica of Horses 136
5. *Acute Dilatation of the Stomach. Dilatatio ventriculi acuta.* 136
   (a) Acute Dilatation of the Stomach in the Horse 136
   (b) Acute Dilatation of the Stomach in Other Animals ... 147
6. Chronic *Dilatation of the* Stomach. Dilatatio ventriculi chronica 149
7. Foreign Bodies in the Stomach. Corpora aliena ventriculi ... 152
   Foreign Bodies in the Stomachs of Other Animals 154
8. Ulcer *of the Stomach.* Ulcus ventriculi 155
9. Neoplasms in the Stomach. Tumores ventriculi 158
10. Torsion of the Stomach. Torsio ventriculi 160
    Other Dislocations of the Stomach 164
11. Hemorrhage from the Stomach. Hemorrhagia ventriculi 165
12. Intestinal Hemorrhage. Enterorrhagia 167
    Rectal Hemorrhage of Cattle 170
13. Acute Intestinal Catarrh. Catarrhus intestinalis acutus 170
14. Acute Gastro-intestinal Catarrh in Young Animals 183

PAGE

15. Catarrhal Intestinal Colic. Enteralgia catarrhalis.......... 187
Abdominal Pain in the Uterine Contractions............. 190
16. Chronic Intestinal Catarrh. Catarrhus intestinalis chronicus. 190
17. Membranous Enteritis. Enteritis membranacea............ 194
Enteritis Pseudomembranacea of Cats.................. 197
18. Gastro-enteritis 197
Enteritis in Fowls...................................... 210
Dysentery in Man.. 211
Meat Poisoning and Botulism in Man.................... 212
19. Bloating of the Intestines. Meteorismus intestinorum....... 213
20. Impaction. Obstipatio..................................... 219
(a) Impaction of the Intestines in the Horse 219
Sand Impaction.................................... 230
(b) Impaction of the Intestines in Carnivora 232
(c) Impaction of the Intestines in Ruminants 235
(d) Impaction of the Intestines in Hogs 237
(e) Impaction of the Intestines in Rabbits 238
(f) Impaction of the Intestines in Fowls 238
21. Internal Displacements of the Intestine. Obturatio intestini. 239
22. Narrowing of the Intestines. Stenosis intestini............. 249
23. Occlusion of the Mesenteric Arteries. Thrombosis et emboli arterium mesenterialum............................ 256
Thrombosis of the Arteria Coeliaca..................... 271
Thrombosis of the Mesenteric Venae.................... 272
24. Internal Strangulation of the Intestine. Incarceratio et strangulatio intestini................................ 272
25. Volvulus and Torsion of the Intestine. Volvulus et torsio intestini 286
26. Intestinal Invagination. Invaginatio intestini.............. 299
27. Colic 307
28. Animal Parasites in the Stomach.......................... 314
(a) Gastrophilus Larvæ in the Stomach of the Horse..... 314
Nematodes in the Stomach of the Horse............ 322
(b) Stomach Worms in Sheep and Goats. Strongylosis ventriculi ovium et caprarum.................... 323
Stomach Worms in Deer.......................... 330
(c) Stomach Worms in Cattle. Strongylosis ventriculi bovum 330
(d) Parasites in the Stomach. Swine 332
(e) Parasites in the Stomachs of Carnivora 334
(f) Parasites in the Stomachs of Rabbits and Hares... 335
(g) Parasites in the Stomachs of Birds................ 335
29. Intestinal Worms. Helminthiasis......................... 338
(A) Tapeworms. Cestodes 342
(a) Tapeworms in the Horse 343
(b) Tapeworms in Cattle 344
(c) Tapeworms in Sheep and Goats.............. 345
(d) Tapeworms in Dogs 346
(e) Tapeworms in Cats 352
(f) Tapeworms in Rabbits 352
(g) Tapeworms in Birds 353
(B) Trematodes 355
(C) Nematodes. Roundworms 356
(a) Ascaridæ in Mammals. Ascariasis............ 356

PAGE
(b) Heterakis Infestation of Birds. Heterakiasis .. 368
(c) Oxyuriasis 371
(d) Trichotrachelidæ 373
(e) Dochmii in the Intestines 374
a. Dochmii in Dogs 374
Dochmii in Cats and Felines 380
Dochmii in Silverfoxes 380
b. Dochmii in Cattle 381
Dochmii in Sheep and Other Animals 385
(f) Palisade Worms in Horses. Sclerostomiasis equorum 386
(g) Oesophagostomes in the Intestines 393
Linguatulosis nodularis 397
(h) Strongyloides in the Intestines 397
(i) Echinorrhynchus Gigas 401
Echinorrhynchi in other animals 403
30. Protozoa in the Intestines. Coccidiosis intestinalis 403
(a) Red Dysentery in Cattle. Dysenteria coccidiosa bovum 403
(b) Intestinal Coccidiosis in Sheep and Goats. Coccidiosis ovium et caprarum 410
(c) Intestinal Coccidiosis in Hogs 412
(d) Intestinal Coccidiosis in Carnivora 413
(e) Intestinal Coccidiosis in Rabbits. Coccidiosis intestinalis cuniculorum 413
Intestinal Coccidiosis in Guinea-pigs 416
(f) Intestinal Coccidiosis in Fowl. Coccidiosis int. avium. 416
(g) Trichomonas in the Intestines of Fowl 419
Amebic Dysentery in Animals 422
Amebae in the Intestines of Other Animals 422
Amebic Dysentery in Man 422
(h) Flagellates and Infusoria in the Intestines 423

SECTION VI.

Diseases of the Liver.

1. Icterus 424
2. Gallstones. Cholelithiasis 428
Foreign Bodies in the Liver and Gall-ducts 431
3. Fatty Liver. Hepar adiposum 432
4. Amyloid Degeneration of Liver. Degen. amyloidea hepatis .. 434
5. Rupture of the Liver. Ruptura hepatis 436
6. Acute Parenchymatous Hepatitis. Hepatitis parenchymatosa acuta 438
Hepatitis parenchymatosa acuta diffusa ovium 439
Hepatitis enzootica porcellorum 441
7. Acute Yellow Atrophy of the Liver. Atrophia hepatis flava .. 441
8. Lupinosis 443
9. Suppurativae Hepatitis. Hepatitis suppurativa 446
10. Nodular Necrosis of Liver. Necrosis nodosa hepatis 448
11. Chronic Interstitial Hepatitis 450

PAGE

12. Neoplasms in the Liver. *Neoplasmata hepatis* 461
   Carcinoma of the Liver. Carcinoma hepatis 461
   Other Neoplasms in the Liver 462
   Tuberculosis and Actinomycosis of the Liver 463
13. Animal Parasites of the Liver 463
   (a) Echinococci in the Liver 463
   (b) Distomatosis. Liver Flukes 468
   Other Distomes in the Liver 491
   (c) Cysticerci in the Liver. Cysticercosis hepatis 491
   (d) Coccida in the Liver. Coccidiosis hepatis 494
   Other Animal Parasites in the Liver 495

SECTION VII.

Diseases of the Pancreas.

Diseases of the Pancreas 497

SECTION VIII.

Diseases of the Peritoneum.

1. Ascites. Hydrops ascites 501
2. Peritonitis 509
3. Tumors of the Peritoneum 518
4. Animal Parasites of the Abdominal Cavity 519

Diseases of the Respiratory Organs.

SECTION I.

Diseases of the Nasal Cavities and of the Accessory Cavities.

1. Hemorrhage from the Nose. Epistaxis 521
   Thrombosis of the Nasal Veins 523
2. Nasal Catarrh 523
   Nasal Catarrh, with Cutaneous Desquamation in the Neighborhood of the Nose, in Horses 528
   Benign Infectious Catarrh of Cattle 529
3. Contagious Nasal Catarrh of Birds. Coryza avium contagiosa 530
4. Infectious Nasal Catarrh in Swine. Rhinitis infectiosa suum 532
5. Contagious Nasal Catarrh of Rabbits. Rhinitis contagiosa cuniculorum 536
   Contagious Nasal Catarrh in Guinea-pigs 539
   Contagious Nasal Catarrh of Cats 539
6. Croupous Rhinitis. Rhinitis crouposa 540
   Diphtheroid Inflammation of the Nasal Mucosa 541
7. *Follicular Inflammation of the Nasal Membranes.* Rhinitis follicularis 541
   Rhinitis Bullosa 542

PAGE

8. Neoplasms in the Nasal Cavities. Tumores narium........ 545
Enzootic Neoplasms in the Region of the Nasal Bone....... 546
9. Animal Parasites in the Nasal Cavities.................... 547
(a) Œstrus ovis.................................... 547
Œtrus Diseases in Other Animals.................. 551
(b) Pentastomum tænioides.......................... 551
Other Parasites in the Nose........................ 554
(c) Nasal Catarrh in Rabbits. Caused by Coccidia. Rhinitis coccidiosa cuniculorum.................... 555
10. Catarrh of the Maxillary Sinus. Catarrhus antri Highmori. 556
Myxoid Degeneration of Turbinated Bones and of the Facial Bones in the Horse.................................. 561
11. Catarrh of the Frontal Sinus. Catarrhus sinus frontalis.... 562
12. Catarrh of the Guttural Pouches. Catarrhus sacci aerophori. 564

SECTION II.

Diseases of the Larynx.

1. *Catarrh of the Larynx. Catarrhus laryngis*................ 569
Epizootic Laryngo-tracheitis of Horses 573
Epizootic Laryngo-tracheitis of Cattle 574
Japp Disease in Lambs................................. 575
2. Croupous Laryngitis. Laryngitis crouposa................ 575
3. *Edema of the Larynx. Oedema glottidis*.................. 579
4. Neoplasms of the Larynx. Tumores laryngis............... 581

SECTION III.

Diseases of the Bronchi.

1. Bronchial Catarrh. Catarrhus bronchialis................. 583
Bronchial Croup...................................... 592
2. *Narrowing of the Large Bronchi. Bronchostenosis*......... 593
3. Asthma Bronchiale...................................... 594
4. Lung-worm Disease. Bronchitis et Broncho-pneumonia verminosa 595
5. Animal Parasites in the Air Passages of Fowl.............. 614
Syngamus 614
The Air Sac Mite of Chickens. Cytodites nudus.......... 616
Laminosioptes cysticola................................ 617
Monostomum flavum..................................... 617

SECTION IV.

Diseases of the Lungs.

1. *Congestion of the Lungs and Edema of the Lungs. Hyperæmia et Oedema pulmonum*.......................... 618
Gas Poisoning.. 622
2. Pulmonary Hemorrhage. Hæmoptoe........................ 624

PAGE

3. Alveolar Emphysema. Emphysæma pulmonum alveolare.... 627
   (a) Acute Alveolar Emphysema.................... 627
   (b) Chronic Alveolar Emphysema. Emphysæma pulmonum alveolare genuinum.................... 629
4. Interstitial Emphysema of the Lungs. Emphysæma pulmonum interstitiale.................... 639
5. Croupous Pneumonia. Pneumonia croupousa.............. 643
6. Catarrhal Pneumonia. Pneumonia catarrhalis.............. 671
   Infectious Bronchitis and Bronchopneumonia in Horses... 678
   Atelectasis of the Lungs.................... 682
7. Enzootic Pneumonia of Young Animals.................... 684
8. Mycotic Pneumonia. Pneumonomycosis.................... 695
9. Purulent Pneumonia. Pneumonia suppurativa.............. 699
10. Pulmonary *Gangrene.* Gangræna pulmonum.............. 701
11. Chronic Interstitial Pneumonia. Pneumonia interstitialis chronica.................... 707
12. Echinococcus in the Lung. Echinococcosis pulmonum....... 710
   Other Parasites of the Lungs.................... 712
13. Neoplasms of the Lungs. Tumores pulmonum............. 713

SECTION V.

Diseases of the Pleura.

1. Inflammation of the Pleura. Pleuritis.................... 715
2. Hydrothorax.................... 732
3. Air in the Pleural Cavity. Pneumothorax................. 733

Diseases of the Circulatory Organs.

SECTION I.

Diseases of the Pericardium.

1. Pericarditis.................... 738
   Compression of the Heart.................... 754
2. Dropsy of the Pericardium.................... 754
   Hemopericardium.................... 755
3. *Pneumopericardium*.................... 756

SECTION II.

Diseases of the Heart.

1. *Palpitation of the Heart*.................... 757
2. Bradycardia.................... 759
3. Intermittent Action of the Heart.................... 766
   Adams-Stoke's Group of Symptoms.................... 768
4. Heart Weakness. Insuffientia cordis.................... 764
   Acute Disturbance of the Circulatory Organs............. 773

		PAGE
5.	Dilatation of the Heart	777
6.	Hypertrophy of the Heart	783
7.	Myocarditis	785
	(a) Acute	78[illegible]
	(b) Chronic	789
	Sequelae of Foot and Mouth Disease	791
	Ossification of the Heart Muscle	792
8.	Fatty Heart	793
9.	Rupture of the Heart	793
10.	Tumors of the Heart	794
11.	Animal Parasites in the Heart	795
12.	Acute Endocarditis	795
13.	Valvular Diseases	802
	(a) Insufficiency of the Aortic Valves	805
	(b) Stenosis of the Aortic Opening	808
	(c) Insufficiency of the Bicuspid or Mitral Valve	808
	(d) Stenosis of the Left Auriculo-Ventricular Opening	810
	(e) Insufficiency of the Pulmonary Valves	810
	(f) Stenosis of the Opening of the Pulmonary Artery	810
	(g) Insufficiency of the Tricuspid Valve	811
	(h) Stenosis of the Right Auriculo-Ventricular Opening	812
	(i) Congenital Heart Lesions	812
	(k) Combined Valvular Lesions	813

SECTION III.

Diseases of the Blood Vessels.

1.	Chronic Induration of the Arterial Wall. Arteriosclerosis	820
2.	*Dilatation of the Aorta. Aneurysma Aortae*	824
	Aneurysms of Other Blood Vessels	827
3.	*Obstruction of the Larger Arteries*	828
	Thrombosis of the Pulmonary Artery	832
4.	*Obstruction of the Large Venous Trunks*	833
5.	Rupture of the Large Blood Vessels	834

Diseases of the Digestive Organs

Section I.

DISEASES OF THE BUCCAL CAVITY.

1. Catarrh of the Buccal Cavity. Stomatitis catarrhalis.

(*Stomatitis Simplex, Stomatitis Erythematosa,* ***Mundkatarrh*** [German].)

Etiology. Catarrh of the buccal mucosa may occur **primarily** after injury by sharp parts of plants, by pointed foreign bodies, such as fragments of bones, nails, etc., also in consequence of faulty teeth, improper bridle bits, by rough examination of the mouth. The buccal cavity of cattle may develop a catarrhal or even an ulcerative inflammation after penetration into the mucosa of licked up hairs or of the bearded grains of barley. (Koiranski). According to Dongall, the plentifully developed glandular hair of the chickpea (Cicer arictinum) may have a similar effect on animals on pasture. According to recent and extensive investigations by Stalfors, the ulcers on the dorsum of the tongue of cattle are at times also caused by hard particles of feed (straw, chaff, beards of grains) operating in conjunction with the peculiar lingual structure and the movements of the tongue while feeding (opening of the dorsal groove of the tongue while protruding the tongue and closing of the same when it is drawn back and moved about in the mouth during mastication). Essentially this is an ordinary traumatic wound caused by particles of feed or hair being pressed into it, which, however, very frequently becomes infected with actinomyces, pus organisms, etc., and may, therefore, become enlarged and more or less malignant. (See Actinomycosis in Volume I.) According to its genesis, this ulcer of the tongue is observed almost exclusively in animals kept on dry feed, sometimes in animals at pasture during dry years. A similar chronic traumatic inflammation of the tongue in swine was described by Joest, in which the injury was brought about by parts of glumes penetrating the mucous membrane, which later also became infected with actinomyces, causing a

thickening in the body of the tongue 5 cm. long presenting flattened, button-like, warty protuberances on the surface, separated by deep furrows which were filled with particles of food (Also in cattle on dry feed one will frequently observe on the gums and on the inner surfaces of the lips, awns and hairs which have penetrated the mucous membrane.) In pigs from 4 to 8 months old the formation of granulation tissue is frequently observed between the upper, and less frequently between the lower, first molar and the wall of the cheek, probably due to mechanical influences and injuries (Kidon-Szlávik).

Scalding by hot feed (distillery slop, broth) or hot medicinal applications may also cause inflammations.

Chemical irritants may likewise be the causative factors particularly such as are applied externally as medicines, and may accidentally be licked up by the animal or otherwise come in contact with the mucosa of the mouth. It sometimes occurs that animals drink of solutions of carbolic acid or corrosive sublimate, prepared and used for purposes of disinfection. Occasionally internal medicines, when used in too strong a concentration, may exert a caustic effect, and this is frequently the case with tartar emetic. Some medicines, like mercury, iodine or lead, may produce a catarrhal affection of the mouth after long continued internal use. This condition may also be produced by strongly fermenting distillery slop, or other fermenting food materials. The milk of animals suffering from diseases of the udder, from foot-and-mouth disease, or from variola, produce inflammation in the mouth of the sucking young. Further causes of catarrhal inflammations are the poisons contained in such plants as spurge, hemlock, mustard, ranunculaceæ, etc., occasionally also Swedish clover and fresh, insufficiently dried hay. Catarrhal inflammation of the mucous membrane of the mouth of dogs has repeatedly been seen when these animals have been running around in fields abounding with stinging nettles. The hairs of the latter get into the mouth on inspiration and penetrate the mucous membrane. (Rohr.)

Feed infected with various fungi is believed to be particularly prone to cause inflammation. However, the experiments of v. Tubeuf have failed to demonstrate the correctness of this claim.

Caterpillars or the hairs of caterpillars mixed with feed cause inflammation both in consequence of traumatic injury and also on account of the presence of acrid poisons; injurious caterpillars are especially Bombyx processionalis and Porthesia chrysorrhœa. Similar effects are produced by plant lice when present in large numbers; mites (Acarus farinæ?) are likewise said to produce such inflammatory processes.

Kriesche found, in a dog and in a horse with depressed appetite, leeches (hirudo) attached to the internal surface of the cheek.

Secondary catarrh of the buccal mucosa starting from the gums or their neighborhood may be due to the eruption or change of the teeth (gingivitis, oulitis).

Catarrh is frequently seen in the course of infectious and intestinal diseases, in which decomposition products of the saliva and the desquamated epithelia accumulating in the buccal cavity undoubtedly play an important rôle. Catarrh of the buccal mucosa is also common in inflammatory conditions of the neighboring organs (pharynx, larynx, salivary glands). A mucous membrane, debilitated by general nutritive disturbances, rachitis, anemia, marasmus, becomes predisposed to the development of catarrhal affections. This is also, partially at least, the reason why disease of the mucosa of the mouth is seen occasionally in dogs during gastro-intestinal inflammations or sepsis.

Finally, stomatitis occurs during the prodromal stage of specific infectious diseases (foot-and-mouth disease, infectious pustular stomatitis of horses, variola, rinderpest, malignant catarrhal fevers, scorbutus, diphtheria, etc.).

Symptoms. In **acute stomatitis**, an increased sensibility of the mucosa of the mouth is observed, and this causes the animal to take and chew its food in a more careful, deliberate manner. Thirst is generally increased. On examination, the mucous membrane exhibits an increase of temperature and congestion; the increased redness may be uniform or in patches; tenacious mucus is present. Sometimes there is some swelling, and in horses it is often considerable on the hard palate (frog on hard palate). This is due to a marked hyperemia caused by compression of the venous plexus. Sometimes the tongue shows edematous swelling with transverse furrows or dental impressions. Occasionally, the excretory ducts of the mucous glands become occluded, and then small grayish nodules (stomatitis follicularis) are seen here and there, particularly on the internal surfaces of the lips. These may exceptionally form small, shallow ulcerations, which generally heal promptly; however, larger ulcers are sometimes found and such may also appear on the skin in the neighborhood of the lips and the nose; the lymphatic glands and their vessels may also become swollen (Kohnhäuser). Vegetable barbs which penetrate the mucosa also occasionally lead to the formation of small ulcers, up to pea size; the vegetable parts which have caused the ulceration may generally be seen at their base (Koiranski). In young horses which are changing their molars, an enzootic alveolar periostitis with uni- or bilateral swelling of the superior maxilla may develop, as in osteoporosis (Payne). In certain cases of stomatitis, an edematous swelling of the lips and cheeks, also a catarrhal inflammation of the nasal mucosa, may be observed. In a colt which had just been weaned, Hasse saw stomatitis with excoriation and soreness of the lip

at the level of the molar teeth; the affection is called "Boca rajada" and might be caused by feeding of sharp, cutting species of Panicum.

The saliva is increased, salivation occurs, and a foamy fluid with large air bubbles adheres to the lips. Owing to the decomposition of the saliva and of the desquamated epithelia, a sweetish disagreeable smell becomes manifest (foetor ex ore).

The loosened epithelia, which are found on the surface of the tongue mixed with particles of food, form a light or dark grayish, occasionally greenish or brown cracked layer, which in carnivora hides the red flesh color of the adjacent muscular layer.

In cattle and in cats, the epithelial covering of the filiform papillæ, which is even normally well developed, proliferates a good deal and the upper surface of the tongue becomes markedly white, or yellowish and rough. A similar, though generally less marked, deposit may also be seen in other parts of the buccal cavity, such as the gums, the internal surface of the cheeks, the corners of the lips.

Berg described stomatitis epithelialis as an epithelial affection on the surface of the tongue, but more frequently at both rounded-off corners of the premaxilla, and presumably caused by bacterial infection. It consists in the appearance of small, round, lemon-yellow, grayish spots on the surface of the tongue, on which the epithelium has become loosened, appearing saturated with a serous fluid and slightly raised from the base. The sharply circumscribed epithelial defects rapidly coalesce. At the same time the process spreads forward as far as the muzzle and the hair on the lips as well as backward, without, however, progressing as far as the mandibles. The disease terminates favorably in a few days and really is only of diagnostic importance in so far as it may be mistaken for healed erosions of foot-and-mouth disease. Because of the absence of foot and udder (teat) lesions and its noncontagious character, it is easily distinguished from this disease. A similar anomaly of the epithelium of the dorsal surface of the tongue has been described by Magnusson and seems to occur quite frequently in Sweden. In this affliction roundish elevations of the epithelium with brown pigmented margins appear on the dorsal surface of the tongue, which remain limited to the superficial horny layer.

According to Berg, erosive stomatitis may be observed in cattle and is probably caused by feeding cake having sharp, hard edges with a simultaneous bacterial infection. There appears on the hard palate and inner surface of the lips, sometimes on the lower surface of the tip of the tongue and on the muzzle, rounded spots, at first pale red, later deeply reddened, somewhat larger than a lentil, which are changed into circular erosions upon the desquamation of the epithelium and heal very rapidly. This disease, which is not infectious, disappears without a trace in from one to two weeks.

The term pip (Pellicula linguæ, Pituitas) designates in birds a tough pseudomembranelike epithelial covering on the dorsal surface of the tongue, which becomes especially well pronounced when its surface becomes dry from breathing through the opened beak.

Chronic stomatitis rarely leads to disturbances of the general health, which are then similar to those of the acute form, but usually milder. The common ulcer of the dorsal surface of the tongue of cattle as a rule does not cause a disturbance of the health of the animal. It is usually discovered in the course of casual examinations, presenting the form of a round, oval or transverse slit-like defect of the epithelium, or, more frequently, of the mucous membrane, with a whitish epithelial

margin and a grayish, brown, or reddened base which is frequently covered with a smeary pus. As a rule awns and particles of food will be found to have penetrated into the base and microscopical examination often reveals actinomyces in the pus adhering to them. The chronic inflammation of the mucous membrane of the cheek in the pig leads to the formation of one or more pointed, fibroid, deposits up to 2 cm. in length, which frequently occupy the space between the mucous membrane of the cheek and the gum of the first molar. By traction on the nerve endings in the young connective tissue while eating, especially when grain is fed, pain is caused, as the result of which the pig will suddenly stop eating and raise its head. Emaciation follows and will become noticeable in a few weeks.

Course. Primary stomatitis always takes a favorable course. After the swelling and the hyperemia subside, the ulcerations which may have been formed, heal with the development of a new epithelial covering. Stomatitis follicularis likewise ends in recovery in one to two weeks, even if neighboring cutaneous parts are involved, although scars may remain if the ulceration has been deep. The course of secondary stomatitis depends upon the nature of the underlying disease.

Diagnosis. The diagnosis of the local catarrhal conditions is not at all difficult, and a careful examination of the other organs makes it possible to determine whether such a stomatitis is a primary affection or the complication of another disease. If stomatitis is due to mycotic invasion, numerous mycelia and spores of the causative mould (Polydesmus exitiosus, Puccinia graminis, etc.,) are often found in the scrapings from the mucous membrane. Since stomatitis occurring in specific infectious diseases (foot-and-mouth disease, stomatitis pustulosa contagiosa) presents the same symptoms as a simple catarrhal stomatitis, it is always necessary to consider the possibility of such causative diseases during the first day of the affection.

Treatment. In most cases, a regulation of diet and the removal of the causative factors, after they have been ascertained suffice to procure a speedy recovery. Whenever there is a stomatitis which is at all severe, no coarse dry feed should be given. Herbivora should receive green feed, fine hay, bran, a mash of bruised grain. Hogs are given bran mixed with whey; carnivora prefer milk, soups or finely chopped meat. A change of feed is particularly indicated when the disease appears to be due to spoiled or otherwise improper material. Fragments, splinters, etc., have to be removed by hand or by the aid of forceps; pointed and sharp margins of teeth must be removed by the proper instruments. The simplest method of local treatment consists in frequently offering the animal fresh water so it may rinse its buccal cavity. To remove the large amount of sticky saliva and the loosened epithelium, the mouth should be

rinsed daily from one to three times with fresh water containing vinegar and table salt (1/8 liter vinegar and 1 tablespoonful salt to 1 liter water) or with water containing 1% to 2% hydrochloric acid. In small animals sage tea containing vinegar and honey (1:1:10) may be used, or water containing hydrochloric acid or camomile infusion, decoction of oak bark, etc. Especially in extreme cases weak solutions of disinfectants may be used, such as potassium chlorate (1% to 3%), potassium permanganate (½%), alum or tannin (½% to 2%), boric acid (3%), hydrogen peroxide solution, 3% perhydrol or hyperol; in small animals also odol (salol 4, alcohol 90, oleum menthæ pip., tincture of vanilla āā 1, tincture of coumarin, sugar āā ½), or quinosol in alcohol and water (1:30:1000). In the treatment of stomatitis follicularis a 1% to 2% solution of creolin or of saponified cresol solution, may also be considered. The affected parts may be cleaned by lightly rubbing them with a sponge or with a piece of linen or oakum or a pledget of cotton fastened to the end of a stick or probe and moistened in a selected solution. A fountain syringe may also be used to rinse out the mouth or it may be syringed out by attaching a piece of rubber tubing or a hard rubber or metal piece to a syringe and introducing this several inches into the mouth at the commissure of the lips and directing the point against the opposite side of the mouth. In case the fore part of the mouth is diseased it is better to direct the point of the syringe toward the front, slowly emptying the syringe, the head of the patient being held downward and somewhat sideways to facilitate the flowing out of the solution which is being flushed about in the mouth by the movements of mastication.

Chronic catarrh is treated by painting the affected parts with a 1% to 2% solution of silver nitrate. Chronic inflammation of the mucous membrane of the cheek in swine will be speedily relieved after severing the fibroid connections between the mucous membranes of the cheek and the gums.

2. Vesicular Inflammations of the Mouth. Stomatitis Vesiculosa.

(*Sporadic aphthæ, sporadische Maulseuche* [German]; *Stomatitis aphthosa sporadica.*)

Vesicular inflammation of the mouth is an inflammatory process distinct from foot-and-mouth disease and from stomatitis pustulosa contagiosa of the horse, leading to the formation of vesicles varying in size and filled with a clear fluid.

Most veterinary authorities describe this disease under the name of stomatitis aphthosa. The authors believe that this is not quite correct because it is inadmissible to designate vesicles as aphthæ. The latter term more properly designates pseudomembranes of the buccal mucosa (see page 9). In distinguishing this affection from foot-and-mouth

disease (aphthæ epizooticæ) it is frequently designated as aphthæ sporadicæ.

Occurrence. The disease has, so far, been observed only in horses and cattle. It generally occurs sporadically (Guittard, Iwersen, Vontobel, Pr. Mil. Vb.), but among horses also enzootically (Dieckerhoff, Bochberg) or even epizootically (Theiler). Epizootics of the disease have been observed particularly in South Africa.

Etiology. Pasturing in rape stubble fields or ingesting much Swedish clover has frequently been held to be the cause of the disease, which was then believed to be due to mycotic invasion (Polydesmus exitiosus, Uromyces occultus). (See also Clover disease.)

Michaelis observed three cases where the disease came on simultaneously with an exanthematous eruption near the feet after feeding on potatoes which had already germinated. Bochberg saw five horses of one owner affected after they had received mouldy and partly dusty feed. Dieckerhoff saw 50 horses out of 100 of the Trakehn Stud become sick successively, and therefore believed that the disease was infectious and that it found its portal of entrance through a lesion of the buccal mucosa. However, since Dieckerhoff's and also Bochberg's inoculation experiments were not successful, they deny the infectious nature of the disease.

Symptoms. In **horses** the buccal mucosa becomes reddened, sensitive and hot and there may occur a slight elevation of temperature, at the same time vesicles appear of the size of a millet seed, lentil or bean, filled with a clear watery or yellowish serous fluid, thin-walled, flattened or even umbilicated. These vesicles are seen on the inner surfaces of the lips, on either side of the lingual ligament, on the gums between the teeth, in the neighborhood of the angles of the mouth, on the lips and on the sides of the tongue. The vesicles burst in three to four days, leaving superficial erosions which remain partly covered for some time by the whitish fragments of the torn wall of the vesicle. Adjoining erosions may become confluent and form irregular defects in the epithelial covering. Vesicular stomatitis occurring in South Africa at this stage exhibits a dirty bluish discolored tongue (hence the name of the disease). The intensely red base of the erosions soon fills up and a new epithelial covering is formed in five to six days, when all traces of inflammation disappear. Bochberg observed the appearance of pustules on the external skin, especially at the angles of the mouth, the *external nares and on the nasal mucosa.*

During the height of the disease salivation becomes quite profuse and persists until healing has taken place. The ingestion of food is more or less interfered with.

In **cattle**, *vesicles appear on the reddened mucosa, particularly on that of the hard palate and of the inner surfaces of*

the lips, but without much salivation; they vary in size from a lentil to a half-dollar piece. They are otherwise like those observed in horses, and the red erosions seen after rupture of the vesicle heal in one to two days and become covered with new epithelium (Vontobel, Iwersen). Sometimes the animals make smacking noises.

Berg sometimes observed a pustulous exanthema appearing simultaneously on the lower lip, the chin and sometimes under the jaw. In some cases he observed an ulceration of the erosions which gradually increased in size, in which case the disease would run its course in about six weeks. In another form of the disease, frequently observed by Berg, dark red, sharply circumscribed spots, few in number, and sometimes showing a few white vesicles the size of a mustard seed, appeared on the hard palate and frequently on the muzzle.

Diagnosis. In horses, the disease may be confounded with stomatitis pustulosa contagiosa, which is, however, characterized by the simultaneous formation of nodules on the mucosa and the integument of the lips and margins of the nares, and by a purulent change of the contents of the vesicles. Nevertheless, it was proven by the cases reported by Bochberg that the two diseases may present very similar symptoms. Infectious vesicular stomatitis in cattle and horses is characterized by the continued appearance of new vesicles, while those which appeared first show extensive desquamation of epithelium, by the formation of relatively extensive erosions and by the transmissibility of the disease from horses to cattle and vice versa.

In ruminants, the disease may easily be mistaken for foot-and-mouth disease. However, the latter always begins with fever (Iwersen has seen occasionally cases of non-contagious vesicular stomatitis with chills), the vesicles are considerably larger: the patients generally make smacking noises. Appetence is suppressed, the feet and the udder show vesicles and the disease is very contagious and can easily be inoculated. A reliable differential diagnosis can only be made after an observation of several days.

Treatment. This follows in general that outlined for simple catarrh of the mouth. Berg used a strong solution of copper sulphate to good advantage. Walther painted the affected parts with a solution of aluminum chlorate (100 gm. to 1 liter of water, to which a little flour and some sirupy substance, such as honey, is added). On account of the possibility of mistaking the affection for some specific infectious disease, the animals should be isolated until cure is effected.

Stomatitis Vesiculosa in Rabbits. According to Sustmann, this disease, known as salivation in rabbits, occurs frequently, especially in young animals and in well-bred "sport" breeds, and is probably caused by mechanical irritation (hard, sharp-pointed feed) or by wet, spoiled, or decomposed feed.

The symptoms begin with diminished appetite and a slowly increas-ng flow of saliva. The saliva is partly rubbed into the hair around he nose with the fore-feet, thus simulating a wet nose as in nasal atarrh in rabbits. Soon there also appear small white papules and esicles on the lips, frequently also on the point and lateral surface of he tongue or on other parts of the buccal cavity. The vesicles of the kin dry up with grayish white scabs which drop off, leaving a normal ppearing skin. In the late stage of the disease diarrhea may set in, hich may, combined with a depressed appetite, cause the death of the nimal as a result of debility (in young animals up to 50%), while therwise the disease often terminates favorably in a few days. The isease should be differentiated from nasal catarrh in rabbits and hinitis due to coccidiosis.

Treatment consists in providing the animal with good, clean bed-ing, dry feed (oats, hay, bread), or cooked feed, oak and willow bark. he use of drugs is superfluous in fresh cases. The mucous membrane f the mouth may be treated as indicated on page 6, or sodium bicar-bonate may be applied to the tongue.

. Aphthous Inflammation of the Mouth. Stomatitis Aphthosa.

Aphthæ sporadicæ, stomatitis aphthosa, stomatitis mycotica, febris aphthosa simplex, stomatitis benigna; stomatite ul-céreuse bénigne [French] [Cadéac].)

Aphthous inflammation of the mouth is an independent dis-ase of either infectious or toxic origin, characterized by des-uamated epithelial masses and a fibrinous exudate formed on reddened base.

Aphthæ in veterinary medicine designate vesicles of the buccal mucosa which are generally filled with a serous fluid, while in human medicine, according to the histologic investigations of Bohn, Schech and Fraenkel, aphthæ are pseudomembranes formed by desquamated fibrin, below which the tissues are comparatively intact. For the sake of clearness and from comparative pathologic considerations this term which is generally used in human medicine is retained, though histologic studies on the part of veterinarians have not come to the knowledge of the authors.

Out of a large group of diseases evidently quite variable as to their etiology, only two groups will be described in detail in the following chapters.

(a) Aphthous Inflammation of the Mouth of the Young.

(Socalled *Maulschwaemmchen, Kahn*, or *Soor der Saeuglinge* [German]; *Stomatite ulcéreuse des agneaux et des chevreaux* [French].)

This contagious disease of sucklings is characterized by the development of pseudomembranous deposits which are, at first, whitish, later grayish or grayish yellow, on a more or less in-flamed buccal mucosa.

Occurrence. The disease usually occurs in the form of an epizootic. Lambs are most frequently affected, more rarely kittens, also sucking calves and foals. The animals generally sicken during lactation, more rarely directly after weaning and when being fed with flour pastes.

Koenig (1855) and Spinola (1858) mention affections under the name of scorbutus of lambs (Gips) or of symptomatic aphthous disease of lambs which probably belong to this group. The same is true of the disease called croupous stomatitis of sucking foals (Hartmann) and of the ulcerative stomatitis of lambs.

Etiology. The epizootic character of the disease suggested its infectious nature, and indeed Delafond succeeded in transferring it artificially. From the pseudomembranes and from the internal organs of sick animals, Besnoit isolated large Gram-positive cocci, arranged in groups of two or in chains of four; cultures of these, when inoculated, produced the disease in lambs and kittens. Whether the pseudomembranes are primarily due to the bacillus necrophorus, the disease therefore representing a true necrobacillosis as indicated by Mohler, has not been proven. However, in its severest form the disease is very similar to necrobacillosis of the buccal mucosa (infection with Bacillus necrophorus). Joest found the **Bacillus pyogenes** as the cause of fibrinous or fibrinopurulent deposits on the mucous membrane of the mouth in sucking lambs. Under certain conditions the paratyphoid bacillus may produce a pseudomembranous stomatitis without simultaneously causing a pseudomembranous pharyngitis. Lesage observed the condition together with an epizootic myocarditis in lambs.

Susceptibility. Marasmus and anemia predispose to the disease, and deficient cleanliness and moist air of the barn indirectly act in the same manner.

Symptoms. The disease begins with reddening and moderate swelling of the mucosa of the mouth, and after one or two days peculiar membranous deposits are formed on the mucosa. Very fine, whitish points appear on different places of the buccal cavity, particularly on the back of the tongue, on its margins, near the lip, on the gums around the teeth, and behind the angles of the mouth, which enlarge to the size of a lentil and keep on growing, becoming thicker and assuming a gray, later a yellowish color; they are surrounded by an intensely reddened halo. It is difficult to remove the pseudomembranes; if it is done, an intensely red, easily bleeding surface appears; the detached membrane is quite friable and looks like tenacious pus or soft cheese. At the margins of the lips small vesicles and later yellowish-brownish crusts are formed, which bleed easily on being touched. (According to Zürn, the pseudomembranes are only formed after rupture of the vesicles.)

In cases with a favorable termination the pseudomembranes

become detached, beginning at the margins, and leave behind intensely red, easily bleeding erosions. These soon become closed by a newly-formed epithelial covering. In serious cases, the number of pseudomembranous spots increase, and those already formed grow and become confluent. This leads to the formation of continuous thick yellowish-brown pseudomembranes which cover the ulcerated mucosa extensively. Bluish-red elevations are also formed, particularly on the gums, which easily bleed. The ulcerative process may extend into the alveoli and lead to loosening of the teeth.

In consequence of the painful condition of the mucosa of the mouth sucking or the taking of food becomes greatly disturbed and finally impossible. There is profuse salivation, and in severe cases a repulsive foetor ex ore. The tissues around the mouth become dark in consequence of adhering dust and dirt. The sick animals become rapidly emaciated, there may be constipation or diarrhea and colicky pains; a catarrhal pneumonia sometimes sets in, and finally the exhausted animals die.

Course. In lambs less than two weeks old, the disease generally takes a very rapid and often fatal course. In older animals, the course is generally more favorable, but fatal cases occur, especially in consequence of complications. The mortality ranges from 8-15-20% in different epizootics; occasionally all of the affected lambs will die (Koenig). The disease also causes losses in consequence of the fact that it disturbs the development of the animals, even if they do not die from it, and it is very difficult to eradicate it from a herd after it has once made its appearance.

Diagnosis. The white or yellowish spots which are formed together with the symptoms of stomatitis and the vesicles on the margins of the lips furnish a very characteristic picture. The presence of vesicles on the margins of the lips, the inflammatory halo around the lips, and the shallow ulcerations distinguish the disease from thrush (Soor), while the absence of vesicles on the mucosa differentiates it from vesicular stomatitis. In severe cases of aphthous stomatitis, ulcerative stomatitis may be thought of; but as such severe cases usually occur side by side with mild cases, the true nature of the disease can readily be recognized.

Treatment. In consideration of the very contagious character of the disease, healthy young animals and their mothers must be separated from the sick lambs. The healthy animals should be kept under observation and all new cases of infection are to be separated promptly. The pens must be subjected to a thorough cleaning and disinfection. Since the sick animals can, in consequence of the painful character of the disease, not easily suck or not at all, they should be fed with milk obtained from

their mothers by milking; or if they are old enough to be weaned they may receive some other suitable liquid food.

The local treatment consists in frequent washing of the mouth with disinfectant fluid, for instance, 4% boracic acid solution, 3-4% solution of chlorate of potash, 1-2% solution of sulphate of copper.

(b) Infectious Papulous Inflammation of the Mouth of Cattle. Stomatitis papulosa.

(*Gutartige Maulseuche* [German] [Ostertag & Bugge]; *Stomatitis follicularis* [Hess]; *Stomatitis papillaris* [Degive].)

Contagious papulous inflammation of the mouth of cattle is an acute specific disease caused by an ultramicroscopic microorganism; it is afebrile and is characterized by the appearance of flat papules on the buccal mucosa and often also on the muzzle.

Since the papules on the buccal mucosa have a certain similarity in their structure with the aphthæ of man, it is proper to consider the disease in this place.

Historical. The etiology of the disease which made its appearance, epizootically, among Bavarian steers in the stockyards of Berlin (Tiarks) has been cleared up by Ostertag & Bugge (1905), who also showed that it was identical with a disease described by Hess in 1899, under the name stomatitis follicularis, and by Degive (1884) as stomatitis papillaris. The sporadic aphthous disease of Deppe (1899) and the infectious stomatitis seen by Haag (1907) in Bavaria likewise appear to be identical with this disease. According to Tiarks (1904), it is common in the Prussian provinces of Posonia and East Prussia. Finally, some of the diseases called pseudo foot-and-mouth disease of cattle (*q. v.*), probably are also identical with this affection.

Etiology. The cause of the disease is an ultramicroscopic microorganism which is found in the pathologic lesions and in the blood of the sick animals.

Pathogenicity. When particles of the pathologically changed mucosa are inoculated into the buccal mucosa of healthy cattle, they produce the disease in its typical form; it can also be produced by subcutaneous and intravenous injections of the blood of sick animals. Filtered blood serum also caused the disease in the experimental animal in one case, but it was not effective in subsequent transfer experiments, although the defibrinated blood of the same animal remained virulent. If the pathologic products are transferred to the nasal, ocular or vaginal mucosa, the disease is not produced. Spontaneous transmission to healthy calves has also been observed. Younger animals are more susceptible than older ones.

How **natural infection** is produced has not yet been shown. The experiments of Ostertag & Bugge make it possible to believe in an inoculation with the food through injuries in the buccal mucosa.

Pathogenesis. The blood vessels are dilated in circumscribed places of the buccal mucosa; this is followed by migration of leucocytes into the lymph spaces in the neighborhood of the blood vessels of the superficial layers of the mucosa, and between the epithelial cells of the stratum germinativum; in consequence of this, the epithelial covering and the papillæ of the mucosa become thickened. In the meantime the epithelial cells become vacuolated; this vacuolation increases toward the surface and a desquamation of epithelia occurs, so that the papillæ remain covered only by the deepest epithelial layers. In the further course of the process, the epithelia covering the elongated papillæ proliferate and form a new covering. If the upper portion of the papillæ has been destroyed, the latter suffer a diminution in size.

Symptoms. *The incubation stage after artificial infection* lasts an average of two weeks, although transitory, but not characteristic, symptoms may sometimes appear after seven to eight days. Flat nodules of from millet seed to lentil size appear on the internal surfaces and on the margins of the lips, also on the hard palate, particularly on its sharp margin, at the corners of the mouth, and on the inferior surface of the tongue, on the internal surface of the cheeks, and also exceptionally on the soft palate. These nodules may become confluent in the further course of development and form larger patches. Similar nodules often are formed in the integument of the muzzle. The nodules and the area surrounding them at first appear intensely reddened, later on they assume a yellowish-gray color and are then surrounded by a red area. They are firmly connected with the underlying surface and cannot be removed easily, whether as pseudomembranes or otherwise. In the further course the epithelial covering desquamates without the preliminary formation of vesicles and central circular depressions are formed, with a granular, frequently excavated, base, which may either show an intensely red or a blackish color, depending upon the pale red or the grayish-black color of the mucosa of the sick animal. While the erosion grows to the size of a bean or even to that of a silver dollar, the base becomes yellowish. After eight days the erosions are closed by a new epithelial covering, but the healed spots may be recognized for as long as one month by their intensely red color and their roughened surface. Recurrences occur frequently and repeatedly, so that the course of the disease may extend over months; however, it always ends in recovery. Other symptoms are usually absent, although in more severe cases there

may be a formation of crusts on the skin, general emaciati‹ and a bad smell from the mouth.

Liénaux noticed in a cow salivation, grating of teeth and diarrhea in addition to the symptoms above described. Post-mortem examination showed erosions and loss of substance along the whole gastro-intestinal tract similar to those in the mouth. Artificial inoculation was not successful in contradistinction to what occurs in contagious papulous inflammation. Hess mentions the occurrence of very small vesicles in the center of nodules which burst soon and attain the size of a millet seed to that of a pea. Reinhardt often saw dirty, grayish-yellow, peculiarly fissured, dry deposits adhering to and projecting above the mucous membrane.

Diagnosis. The presence of the peculiar flat, nodule-like formations on the buccal mucosa, and occasionally on the muzzle, without disturbance of the general health and without involvement of the feet, the slight extension of the lesions, their eaten-out appearance, with only superficial loss of substance sufficiently characterize the disease. It may be distinguished from vesicular stomatitis by the absence of vesicles in the mouth, and by the presence of the characteristic nodules in the neighborhood of the existing erosions; besides there is increased secretion of saliva in vesicular stomatitis. The same differential features also distinguish the affection from foot-and-mouth disease in which large vesicles are often seen on the back of the tongue or on the feet. In aphthous stomatitis of sucklings, pseudomembranous deposits are developed on the usually reddened tongue and the disease affects only sucklings and young animals which have just been weaned.

Treatment. This is similar to that employed in vesicular stomatitis. Considering the contagious nature of the disease, sick animals should be separated from the healthy ones.

Other Diseases Similar to Stomatitis. Aside from aphthous stomatitis of sucklings and from contagious papulous stomatitis, diseases have been observed in cattle and exceptionally in sheep which, on the basis of pathologic-anatomical changes, are best classified with aphthous stomatitis. Among them are to be mentioned the following: sporadic stomatitis (Maulseuche, Utz), affections similar to foot-and-mouth disease (Bang, Stribolt, Andersen, Kern), pseudo mouth-disease or stomatitis erosiva (Pusch), thrush of cattle (Hajnal), epizootic inflammation of the mouth of sheep (Vigadi), stomatitis ulcerosa in cattle (Bedel), pseudo foot-and-mouth disease (Kantorowicz), stomatitis mycotica (Mohler), stomatitis pseudo-aphthosa (Cadéac).

Concerning their **etiology**, these affections may be divided into three groups. The first group comprises those diseases which are contagious and which consequently must be due to an infectious agency, which in part of these cases may be the bacillus necrophorus (see Necrobacillosis). In the affections of the second group (Utz, Bruemmer, Bang, Stribolt, Andersen, Kantorowicz) injurious food appears to be the exciting cause. The appearance of such diseases has followed upon feeding with green clover. They are not contagious. In the other non-contagious diseases of this kind, nothing positive is known as to their cause. Pusch believes that they may be due to some noxious agent which

...disease producer only when the resistance of the organism has been lowered by overexertion (as in railroad transit), irregular care or sudden change in feed. In this form the disease occurs especially in late summer or early autumn of dry years and as a rule attacks only 10 to 15% of the animals in a flock. Frequently molds are incriminated, but it has not yet been satisfactorily demonstrated that they are capable of producing the disease.

It is not impossible that some of the forms here enumerated belong to the contagious papulous form of stomatitis (see page 12).

As to the **clinical picture,** the aphthouslike diseases may, for convenience sake, be divided into two groups. In one group the buccal mucosa only or possibly also the integument of the muzzle is involved

Fig. 1. Pseudoaphthous stomatitis in a cow. Erosions on the hard palate, cheeks and lips, and on the edges of the lips.

The mucosa of the hard palate, the gums, more rarely the sides and the tip of the tongue, the inner surface of the lips and of the cheeks, present grayish-white to grayish-brown pseudomembranous deposits on a more or less reddened surface, or in other cases, round, intensely red erosions up to the size of a silver quarter, the base of which is partly covered with a grayish-red, or yellowish, thin, flaky or thicker deposit. In some cases one also sees numerous pinhead hemorrhages in the papillæ of the

mucosa. In some animals the muzzle shows lenticular prominences an erosions of the epithelial covering (Fig. 2). Only exceptionally doe the disease assume a febrile course and lead to salivation and disturl ances in the ingestion of food.

The clinical picture of the second group is characterized by tl simultaneous affection of the buccal mucosa, of the integument of tl muzzle, of the integument of the extremities, and of the udder. With a elevation of temperature, the appetite is diminished or entirely lackin; Profuse salivation appears and smacking noises are heard just as i foot-and-mouth disease. The mucosa of the mouth is intensely reddene and covered with corrugated, diffuse, pseudomembranous deposits, esp cially on the hard palate, the gums, the inner surfaces of the lips. Tl pseudomembranes come off in shreds and the erosions heal in a fe days. Lenticular yellowish-red or grayish nodules or crusts with a cei

Fig. 2. Pseudoaphthous inflammation of the mouth in the cow. Epithelial defects and clefting of the epithelium on the dental pad, the upper lip and the muzzle.

tral depression appear on the muzzle and exceptionally on the nasal mucosa. Similar nodules sometimes appear on the mucosa of the mouth The lower extremities show evidences of a dermatitis which is similar to that seen in clover diseases (see under this heading); on the skin of the udder, nodules of the size of a lentil with several vesicles and subsequent crust formation were seen by Kantorowicz.

The disease always takes a favorable course either within a week, or in the variety last described within two to three weeks, slight emaciation having become manifest in the meanwhile.

The **differential diagnosis** has to consider foot-and-mouth disease. It is distinguished by the simultaneous vesicular eruption at the ends of the extremities and on the buccai mucosa. the involvement of the back of the tongue and the absence of the peculiar pseudomembranes on the mucous membranes of the mouth. In the forms of stomatitis here described, the contagious character is usually absent, also the involvement of the extremities and of the udder

Treatment becomes necessary only in the more severe forms with dermatitis at the extremities. The principles of the treatment are the same as those in catarrhal stomatitis (see page 5). Most cases end in recovery without any treatment at all.

4. Thrush. Stomatitis oidica.

(*Soorkrankheit*, *Schwämmchen* [German]; *Muguet* [French].)

The term thrush or soor is applied to patches on the mucous membranes of mouth and fauces, or of the crop in birds, caused by the mycelium of the Oidium albicans.

Historical. The Oidium albicans was discovered by Langenbeck in 1839, and was studied by Berg and Gruby (1842), then by Grawitz and particularly by Plaut (1887), Linossier & Roux (1889), Brebeck & Fischer (1894) and Vuillemin (1898). The numerous investigations have not succeeded in establishing its position among the fungi. Grawitz declared it to be a species of mycoderma; Reess classes it with the saccharomycetes, Robin with the Oidia. Plaut believes it to be identical with certain Monilia. Vuillemin demonstrated askospores in some of the soor-fungi and therefore classes them with the Endomycetes. Castellani does not consider thrush as an independent disease caused by a single organism, but as a group of clinically similar diseases caused by organisms of different species, genera and families (Monilia and Oidia, Endomyces, Saccharomyces and Hemispora) and appearing clinically as white and yellow thrush.

Distribution. The soor disease is a rare affection in domestic animals and was observed in chickens (Eberth, Reimann, Zürn), in pigeons (Zürn, Klee) and in a turkey hen (Martin), also in an artificially fed pig (Poenaru). According to Zürn, Neveu-Lemaire and Plaut, it occurs in sucking calves and colts. Grawitz has produced thrush experimentally in young dogs.

Etiology. The fungus of soor (Oidium s. Saccharomyces s. Endomyces albicans. Monilia candida) grows in deposits in the form of threads of variable length and thickness, with double curves and enclosures of droplets, granules and vacuoles. The threads are divided by transverse partitions into numerous elongated cells with rounded ends; branched forms occur. In addition to threads, oval yeast cells are seen, 5 to 6 μ long and 4 μ wide, which correspond perfectly with true yeast cells (Fig. 3) and are formed rapidly, especially in the presence of oxygen. The fungus is Gram-positive.

Cultivation. The fungus thrives better on solid than on liquid. acid culture media. Plate cultures are round, waxy, finely granular on the surface; in their depth they show irregular outlines with thready excrescences arranged radially. On gelatine and agar, the cultures are white, on beet or beer-wort gelatine the color is reddish. The best growth is seen on turnips or on carrots in the form of white deposits with uneven surface. The cultures give off a pleasant acid-alcohol like odor. The fungus hardly grows on saliva, but does well on saliva medium; its power of fermentation is slight.

In addition to the soor-threads and yeast cells, the formation of chlamydo spores may be observed by constriction of rounded cells at the ends of the threads; in some forms, askospores develop in sac-like formations (askus).

Natural infection takes place by the localization of the soor fungus, which probably is distributed widely as a saprophyte, if the conditions for it are favorable; for instance, on the ingestion of foodstuffs which ferment readily, in catarrhal disease of the mucous membrane of mouth or crop, in conditions of insufficient nutrition or in the course of debilitating diseases, particularly in young animals. The infection is favored by staying in damp, p o o r l y ventilated places. Direct infection by contact with affected animals does not appear to take place.

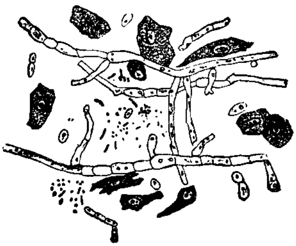

Fig. 3. Microscopical preparation of a thrushy membrane from the oesophagus. Mycelia, oval spores, pavement epithelia, micrococci and bacteria. (After Biedert.)

Artificial transmission with the aid of slight scarification of the mucous membrane is not only regularly successful upon the mucous membrane of the crop in birds, but sometimes also upon the mucous membrane of the mouth in dogs (Grawitz, Haubner) and of the calf (Haubner). Furthermore, the transmission is successful upon the cornea (Fischer and Brebeck) and the mucous membrane of the vagina in rabbits (Stoss), as well as by intravenous injection, in which case it always appears in the form of a generalized mycosis in this species.

Symptoms. The characteristics of thrush are grayish-white or yellowish, later on brownish points, spots or even larger pseudomembranes adhering to the underlying tissue, which does, however, not show any signs of inflammation. Poenaru saw, in a pig, disturbances of deglutition and the mouth was held open. Similar pseudomembranes are found in the pharynx and in the crop; when this occurs, the birds are listless

and perish in spite of good appetite. (See catarrh of the crop.) In the deposits, and also in the mucus from mouth or beak, the characteristic fragments of the fungi are seen on microscopical examination, also leucocytes, epithelial cells and bacteria.

In the observations made by Schlegel the sick chickens disclosed twisted neck, hoarseness, an accumulation of mucus in the mouth and refused feed. The mucous membrane of the beak and of the mouth cavity showed a brown to grayish red discoloration, while the true thrush mycosis was observed only in the glandular portion of the stomach and on the edges of the cuticula of the glandular portion in the form of grayish-brown, disintegrating ulcer-like foci, which did not produce an inflammatory reaction in the surrounding parts. The thrush foci appear in the glandular portion of the stomach at the point where it merges into the muscular portion, and in the first portion of the small intestines as grayish-white to bloody brownish-red, mushy deposits, invading all of the mucous membrane and forming ulcers in the necrotic masses. The affected birds show inappetence and weakness.

Diagnosis. Thrush is distinguished from aphthous and other inflammatory forms of stomatitis by the absence of an inflammatory reaction in the neighborhood of the lesions. It is necessary to demonstrate the typical fungi microscopically before an absolute diagnosis can be made.

(The agglutination and complement-fixation tests gave *positive results. Similar positive results* were obtained when sporotrichum was used as antigen.)

Treatment. The mouth must be kept clean and it may be painted with a 2.5% solution of borax, or, according to Klee, with a solution of bichloride of mercury (1:1000). If the latter solution is employed, great care is required. If the crop is involved, it should be irrigated with a 2% boracic acid solution. (See catarrh of the crop.)

The places where the fowls are kept should be washed with bisulphate of calcium; the feeding vessels should be disinfected by scalding. In this manner a spread of the disease may be prevented.

5. Phlegmonous Inflammation of the Mouth. Stomatitis phlegmonosa.

(*Stomatitis erysipelatosa, S. erosiva.*)

Etiology. After the ingestion of large masses of food contaminated with rust and other fungi, a severe inflammation of the buccal mucosa is occasionally seen in horses and exceptionally in sheep, complicated by suppuration and necrosis of the submucous connective tissue. De Hahn & Hoogkammer saw in East Indian horses an inflammation of the skin of the face, complicated with erysipelatous stomatitis, caused by fungi (hyphomycosis destruens equi). (See Vol. I.) Food which contains many caterpillars or their hairs (porthesia chrysor-

rhea, bombyx processionalis) may act likewise. Lüthens and Köster have shown experimentally that otherwise good food, but mixed with procession caterpillars or their hairs spread on oak leaves, was able to produce a severe stomatitis in horses.

Irritants, such as lye, acids, carbolic acid, croton oil, and scalding of the mouth, are likewise able to produce severe stomatitis. Traumatic lesions of the mucosa may give rise to inflammatory processes of the deeper tissues.

The disease may appear secondarily after purulent or gangrenous processes of neighboring organs (purulent inflammation of the entrance of the esophagus) or in connection with infectious diseases, such as strangles, cattle plague, anthrax, hog cholera, morbus maculosus (purpura hemorrhagica), malignant catarrhal fever, diphtheria of fowl or calves, dog typhus. Erysipelatous inflammation of the tongue forms part of the picture of the disease known as blue-tongue of sheep in South Africa.

Symptoms. The disease sets in with distinctly inflammatory phenomena, great tenderness, intense reddening and swelling of the mucosa; these are generally accompanied by an elevation of temperature. The swelling later on increases still more, and external inspection shows particularly swelling of the lips and of the neighboring portions of the cheeks. The swollen lower lip has fallen and a profuse saliva drips in long threads from the corners of the mouth. The swelling of the tongue is sometimes so extensive that its tip protrudes between the incisors and the organ shows on its lateral margins the impressions of the molars, and on the tip the impressions of the incisors; it is, at the same time, tense and hard.

In the further course, symptoms of pharyngitis make their appearance, and interference with the ingestion of food and water may become complete. The nasal mucosa may also become involved and then a mucous yellowish-brown nasal discharge appears. The submaxillary and the peripharyngeal lymphatics become swollen, hard and painful.

At this stage, the disease has, in most cases, reached its height and recovery sets in slowly. In the severe cases, however, pale yellowish, mushy deposits and pustules appear on the swollen mucosa up to the size of a pea (observed by Bertsche in sheep) and these again lead to the formation of irregular, angry-looking ulcers. Occasionally abscess formation occurs with ichorous, dirty, ill-smelling pus, mixed with fragments of necrotic tissue and parts of food stuffs. In these cases there is fever, listlessness, and indican appears in the urine.

Course and Prognosis. The primary form of phlegmonons stomatitis generally takes a favorable course. If there is no necrosis of tissue and no abscess formation, complete recovery

usually takes place within two or three weeks; even in the severest cases recovery may occur under the proper surgical treatment. Exceptionally, however, the inflammatory process spreads to the larynx, the posterior wall of the pharynx and along the trachea, which then usually leads to an unfavorable termination. The latter also occurs in secondary stomatitis, especially if it occurs in connection with anthrax, hemorrhagic septicemia or with *malignant edema, gas gangrene*, **blackleg** and other malignant diseases.

Treatment. If the inflammation has been caused by noxious food, a change of the latter and of pasture must be instituted. Besides, irrigations and mouth washes, as generally used in stomatitis, are indicated; purulent and ichorous inflammation requires proper surgical interference.

6. Ulcerative Inflammation of the Buccal Cavity. Stomatitis ulcerosa.

(*Mundfaeule* [German]; *Stomacace.*)

Ulcerative stomatitis is especially a disease of carnivora, consisting in an acute inflammation of the gums, which soon leads to necrosis and ulcerative destruction.

Severe forms of stomatitis of herbivora caused by spoiled food, including aphthous stomatitis, are included in this affection by some authors (Friedberger & Fröhner, Gips, Cadéac). But their clinical picture is so different from that of stomatitis ulcerosa, that such a classification does not appear justified.

Etiology. Stomatitis ulcerosa does not represent an independent disease. Although it is in the end caused by micro-organisms under the simultaneous influence of favorable conditions, yet the infectious agent and the favorable influences are not always the same. Especially bacteria normally present in the mouth, such as the **Bacillus salivarius septicus** (found by Fiocca in dogs) and **Staphylococcus pyogenes aureus,** frequently also the necrosis bacillus and other micro-organisms, probably play a part if the favorable influences permit their penetration of the mucous membrane of the mouth. In the primary ulcerative stomatitis in man which frequently occurs epidemically, fusiform bacilli and spirillæ are regularly found, and therefore they are often considered as the probable cause of the disease.

Ulcerative stomatitis of domestic animals develops essentially as a concurrent disease, especially and frequently in old dogs in conjunction with odontoliths, where the irritation due to constant pressure of the latter, and at times also to carious teeth, cause the gums to become more receptive to the penetration of micro-organisms of reduced pathogenicity. Especially

in the more delicate breeds of dogs and cats a decrease in the resistance of the mucous membrane may be caused by debility as also by anemia resulting from insufficient and improper feeding and unsanitary surroundings as well as from severe rachitis and distemper. The disease occurred enzootically in a flock of sheep at the end of the pasturing season without the cause having been determined. Hirsch observed it in kids and Smythe in chickens.

Exceptionally a stomatitis which is primarily benign may pass over into an ulcerative inflammation in consequence of secondary specific infection. The disease also appears as a part of the clinical picture of the infrequent scorbutus in dogs and swine and of chronic hog erysipelas. (See Vol. I.)

The disease is similar in its symptoms to inflammation of the mouth seen in connection with mercurial poisoning after repeated inunctions with gray ointment or after excessive injections of corrosive sublimate (stomatitis mercurialis). (According to Cadéac's experimental investigations, the real cause of this affection is claimed to be the bacillus necrophorus.) Sometimes other agencies like lead, copper or phosphorus may have a similar effect.

Symptoms. The inflammation begins almost without exception at the margins of the gums, around the neck of the incisors of canines, especially in the spaces between the individual teeth. The gums of the molars usually become affected later. The gums at the affected places appear dark or sometimes bluish-red, swollen, very painful, and on being touched bleed but slightly. After one or two days, the free margins of the gums become changed into a pale yellowish or dirty-greenish smeary, mushy mass, which can easily be removed; the live tissue in their neighborhood looks bluish-red and swollen. After the expulsion of the mushy masses, one sees, around the neck of the teeth, ulcers with margins and bases covered by necrotic, discolored detritus. The erstwhile round ulcers become confluent forming an ulcerated discolored surface; the necks of the teeth are left uncovered. After the process has spread into the alveoli, the teeth become loosened and can be extracted easily, or they may fall out spontaneously.

In the further course similar ulcers are formed on the mucosa of the lips and cheeks; these ulcers are found opposite the primary ones on the gums and they are probably due to contact infection. The lips and the cheeks become swollen. In very severe cases the inflammation may extend into the maxillæ and cause necrosis of portions of the bone.

There is always an extremely disagreeable, repulsive, sweetish, cadaverous smell present, emanating from the buccal cavity (foetor ex ore), which pervades the whole space in which the sick animal is kept. There are also symptoms of acute ca-

tarrh of the mouth, especially profuse salivation. The saliva is fetid, *mixed with necrotic shreds and also with blood.* The submaxillary and the cervical glands are acutely swollen and the inflammation extends also to the salivary glands. The increased tenderness disturbs mastication, and the animals either take only fluid food or they swallow soft pieces of meat without chewing.

Later on the symptoms of septic infection develop, such as *febrile elevation of temperature, rapid small pulse, diarrhea,* prostration; the rapidly emaciating animals soon succumb.

Mercurial stomatitis manifests itself, aside from the usual symptoms of mercurialism (eczema of the thin parts of the skin, bronchial catarrh and even bronchial pneumonia, catarrh of the large intestines accompanied by fetid diarrhea, excitement, nephritis and probably petechial hemorrhages of the mucous membrane) in severe salivation, loosening of the teeth, reddening and swelling of the blackened gums (*according to Joest this blackening is due to mercuric sulphide*), which bleed easily when touched and in severe cases become necrotic. The mucous membrane of the lips, cheeks and tongue may later become involved. In stomatitis produced by lead poisoning the edges of the gum disclose a grayish-black discoloration as the result of the precipitation of lead sulphide (lead line) and may also become necrotic and present the formation of ulcers, but at the same time other symptoms of chronic lead poisoning (emaciation, tremor, nervous excitement, paralysis of the larynx, colic, constipation, tense pulse) are present.

Ulcerative stomatitis as a sequel to chronic swine erysipelas manifests itself by hemorrhages and ulcers in the buccal cavity and in the skin, by skin necrosis, chronic swelling of the jaws and emaciation a few weeks after the termination of an acute attack of the disease. For the symptoms of stomatitis ulcerosa in scurvy see Volume 3.

Course. The disease takes a favorable course under favorable hygienic conditions, with the proper treatment and if the patients are still in good condition of nutrition. The inflammatory and necrotic processes become limited, the necrotic portions are shed and healthy granulations form from the margins and from the base of the ulcers. The disease ends then in *complete recovery within one to two weeks. If the cases are neglected,* the animals become much emaciated and a general infection or exceptionally an aspiration pneumonia with pulmonary gangrene develops. (Müller has observed this course in a dog, the authors a similar case in a lion.)

Diagnosis. Since ulcerating stomatitis may form an accompanying symptom of scurvy, the other symptoms of this disease must be *looked for; the absence of hemorrhage indicates* that the stomatitis occurs independently. In young pigs chronic erysipelas must be excluded; only the history and the accompanying conditions can usually give information as to whether the ulceration is due to poisoning.

Treatment. Next to washing and irrigation of the buccal cavity with solutions already mentioned (page 5), careful cauterization of the ulcers with nitrate of silver is indicated. Crusts form on the cauterized places and, after the shedding of the former, clean granulating surfaces appear. Painting

with the following solutions also gives favorable results: 1-2% solution of nitrate of silver, 2-3% solution of chloride of zinc, pure turpentine, 5-10% solution of tannic acid, glycerine, 3% solution of creolin; deodorizing solutions may be used in addition, such as 0.3% solution of potassium permanganate. After the ulcers have become clean, mild disinfectant and astringent solutions are indicated.

During treatment, the animals have to be fed suitably with liquid, juicy nourishment (milk, meat). The teeth which have already become loosened must be removed, a procedure which in the beginning of the disease occasionally leads to a rapid termination of the pathologic process.

In stomatitis as a sequel to poisoning the treatment must at the same time be directed against the respective poisoning.

Diphtheroid Inflammations of the Oral Mucous Membranes. Diphtheritic inflammation of the oral mucosa often develops secondarily in the course of swine plague, diphtheria of calves, and the diphtheria of grown cattle which is essentially the same disease; also in malignant catarrhal fever, in rinderpest, in severe cases of aphthous and pseudo-aphthous stomatitis. In addition, an independent diphtheritic necrosis of the oral mucosa occurs which probably originates through the influence of the bacillus of necrosis, a bacterium which lives as a saprophyte in the mouths of healthy animals, and also in barns and stables, and the localization and multiplication of which may become possible under the influence of debility, nutritive disturbances, local injuries to the tissues, or bad hygienic conditions. Fuchs observed an enzootic in young cattle, in which paratyphoid bacilli were isolated.

Joest found **Bacillus pyogenes** in the yellowish, smeary masses on the mucous membrane of the mouth and pharynx in young lambs in a flock where the ewes were suffering from a necrotic inflammation of the udder due to this bacillus (see page 10). In a case described by Froehner in a horse the diphtheroid stomatitis was caused by drenching with a too concentrated mixture of chloral hydrate (chloral hydrate 25, extract of aloes 20, ether and alcohol āā 75).

Sporadic cases of the disease are noted occasionally in all species of mammalians, especially in cachectic conditions, in the course of protracted gastroenteritis or of a protracted colic in horses, also following upon rachitis and osteomalacia, and accidentally after injury of the mucous membranes of the mouth. In enzootic distribution it has been observed in lambs (Rivolta), calves (Fuchs), goats (Stallmann), particularly in young pigs two to three weeks old (Bang, Lauritsen, Mathis); in the latter it is apt to appear during the winter months when the stables are dark and cold and are not ventilated. Excessive feeding of the mother animals with flour seems to promote the infection. Wyssmann saw cases in combination with diphtheroid pharyngitis; Draegert observed in young cattle, concurrently with diphtheroid deposits on the mucous membrane of the fore-stomachs, hemorrhages of the abomasum and intestines and occasionally with sharply defined rosette-like necrotic foci in the liver varying in size from that of a hazelnut to that of an apple, resulting from the accumulation of **Bacillus necrophorus** in the diseased tissue.

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Symptoms. The clinical picture is variable. In some cases yellow, dry, closely adherent deposits of varying thickness form in spots on the *mucous membrane*, and are exfoliated gradually. In others a yellowish or whitish soft caseous layer forms quite rapidly, it is discharged promptly and then leaves ulcers which are more or less deep. In the cases reported by Giovanoli in goats there appeared on somewhat raised areas yellowish vesicles up to the size of a pea, which upon bursting left flat ulcers with a reddened base and yellowish edges, followed by scab formation and excessive proliferation of the involved tissues. Similar observations were made by Goddard in lambs and yearlings pastured on a stubble field, sometimes in the presence of adult sheep suffering from necrophorous infection of the lips. At the same time the appetite is suppressed and severe general symptoms and fever may also be present.

In enzootic diphtheroid inflammations of young pigs there is observed, aside from the diphtheroid deposits on the inner surface of the lips and the mucous membrane of the mouth, painful swelling of the adjacent skin, emaciation, and sometimes a diphtheroid inflammation of the nasal septum as well as symptoms of a severe pharyngitis. Following the practice prevalent in some places of nipping the corner teeth in suckling pigs, there appears, on either the upper or lower jaw, or on both, a circumscribed, yellow-gray, rather dry deep necrosis as a result of the penetration of **Bacillus necrophorus** into the pulp cavity, and this may *later perforate into the nasal or mandibular cavity or the skin* (Joest). Stallmann observed diphtheroid pseudomembranes, slobbering, disturbance of deglutition, suppressed appetite and fever in goats. Most of the animals died. In young cattle Draegert observed the following: Continued recumbency, frequent gritting of the teeth, lacrimation, nasal discharge, spotted reddening of the mucous membrane of the mouth, of the cone-shaped papillæ and of the palate, with grayish-yellow deposits up to the size of a pea and larger which after dropping off left erosions up to 4 mm. in depth. Sometimes bloody diarrhea was present.

The treatment consists in scraping out the necrotic tissue and washing with astringent solutions, or painting with diluted tincture of iodine. The hygienic conditions must be improved. Internally a mixture of creosote and codliver oil (Ol. jecoris aselli 50 gm., creosoti 1 gm.) may be given in tablespoon doses (Hasenkamp). In the enzootic described by Fuchs, which was caused by paratyphoid bacilli, the disease was arrested after injecting the healthy animals with paratyphoid serum.

Stomatitis gangraenosa (Noma). Gangrenous stomatitis is characterized by rapidly spreading necrosis and putrid decomposition of the mucous membrane, especially that of the cheeks, less frequently that of the gums or the lips, causing extensive destruction of the soft parts or even of the bone.

This very rare condition generally appears as a concurrent disease of ulcerous, aphthous, pseudoaphthous and diphtheroid stomatitis, and in the course of debilitative diseases, as, for instance, following piroplasmosis in dogs (Carpano), in persistent gastroenteritis, dog distemper, chronic nephritis in dogs and chronic bronchitis in cats (own observations). Exceptionally it may appear independently in connection with very meager and irrational feeding and seemingly as an infectious disease (observed by Rivolta in lambs and by Foster in dogs).

The true cause is undoubtedly to be sought in microorganisms. Rivolta found **Bacterium subtile agnorum** in lambs, and Foster protozoa in dogs (stomatitis

protozoica) with secondary staphylococcus invasion. Caprano found in dogs fusiform bacilli in symbiosis with spirochaetes which are also in man frequently looked upon as the cause of pseudomembranous and ulcerous processes in the mouth and pharynx (Vincent's angina). Phillips and Berry observed in a young Spanish dog following distemper a true gangrenous stomatitis, with the presence of **Bacillus fusiformis** and spirillæ in equal number in the necrotic scab, in which **Bacillus fusiformis** later predominated. The gangrenous stomatitis in young calves described by Lengler is caused by **Bacillus necrophorus** (calf diphtheria).

The symptoms differ from those of diphtheroid stomatitis in so far as vesicles up to the size of a dime may or may not appear on the affected parts of the mucous membrane (Rivolta) and later rupture. In either case the affected areas are changed into one or more ulcers covered with discolored, yellowish-gray, smeary material. At the same time a very offensive odor emanates from the mouth. These gangrenous ulcers spread rapidly in all directions, may penetrate to the depth of the skin, and after previously forming hard infiltrations may perforate the wall of the cheeks and lips or spread to the tongue and the jawbone. At the same time severe general symptoms develop which usually terminate fatally.

Treatment is similar to that outlined for diphtheroid stomatitis, but cauterization of the gangrenous tissue may also be considered.

SECTION II.

DISEASES OF THE SALIVARY GLANDS.

1. Salivation. Ptyalism.

(*Speichelfluss* [German]; *Salivatio, Sialorrhoë.*)

Etiology. Salivation is almost without exception a purely secondary symptom, seen most frequently in inflammation of the mucosa of the mouth and pharynx and due to a reflex irritation of the nerves which regulate the activity of the salivary glands. It is also a frequent, though not constant, symptom of parenchymatous inflammation of the salivary glands. Certain chemicals, especially mercury, pilocarpine, arecoline, nux vomica, more rarely arsenic, iodine, lead and copper likewise increase the secretion of saliva; also acrid substances and food contaminated with rust and other fungi. Disturbances of deglutition also are accompanied by salivation; this is especially seen in pharyngitis, in spasm and paralysis of the larynx, and in diseases of the esophagus. Diseases of the gastric mucosa (ulcer, catarrh, inflammation) may cause salivation by reflex irritation. Whether certain diseases of the sexual organs stand in a causal relation to salivation is a point not yet settled in veterinary literature. Anacker reports the interesting observation that a horse always showed salivation when taken to the knacker's yard.

According to Doeve, slobbering and masticatory movements are regularly observed in dogs and cats following the instillation of atropin into the conjuctival sac, as a result of local irritation of the pharyngeal mucous membrane by the atropin as it flows down the lachrymal duct and the nasal cavity. (For the so-called salivation in rabbits, see page 8.)

Symptoms. Saliva flows abundantly out of the mouth, particularly at the corners, either as foam or in long strings, and contaminates the food and other nearby objects. Long-continued salivation finally leads to emaciation. Aside from these symptoms, the primary cause can generally be recognized.

Treatment. Apparently, primary cases of salivation may be treated by the subcutaneous injection of atropine (for large

animals, 0.05-0.1 gm.; for small ones, 0.005-0.02 gm.) or scopolamine (0.01 or 0.002 gm.). This leads, however, usually only to temporary relief. In a case of Dieckerhoff's, continued treatment with arsenic (horses, 0.05-0.8 gm.) led to permanent improvement, while Zündel was successful in the treatment of a cow with iodide of potash. In most cases, the treatment must be directed against the underlying primary condition and no direct treatment of the salivation is required. Diem obtained recovery in a case due to swelling of the parotid gland by repeated injections of pilocarpine, followed by inunctions with an iodine-iodide of potash ointment.

2. Inflammation of the Parotid Gland. Parotitis.

Etiology. Parotitis occurs, as a primary affection, with comparative frequency among domestic animals, after traumatic injury of the parotid region. Aruch observed the penetration of awns (oats, **Hordeum silvaticum**) into the duct of the gland (in one case he counted about a hundred awns of **Hordeum**) as a frequent cause of purulent inflammation in horses. Sometimes parts of **Bromus maximus** (Renault, Mayer) and **Vicia tenuifolia** (Labat) are found in the duct. These and other parts of plants probably take a part in the production of the inflammatory process only in so far as they are carriers of micro-organisms. Parotitis seldom occurs enzootically as a specific infectious disease in cattle, horses, dogs, cats and goats, and is probably caused by a specific microorganism.

Bissauge has repeatedly seen parotitis as an epizootic among cattle n the neighborhood of Orléans in France and always simultaneously vith mumps among children. In a dog a case occurred likewise during ι mumps epidemic among children, and the affection spread to another log. A diplococcus was obtained in pure culture from Stenon's duct (Busquet & Boudeaud). The infection of a dog from sick children was demonstrated beyond doubt by Prietsch.

Parotitis often occurs simultaneously with pharyngitis Calculi frequently cause a catarrh of the duct of Steno, which may be followed by an inflammation of the gland tissue. As a symptom of a specific infectious disease parotitis is occasionally observed in the course of distemper in horses. Sometimes it occurs metastatically in the course of distemper in dogs and infectious pleuropneumonia of horses.

Chronic parotitis develops after repeated traumatic injuries (particularly in race horses) in the presence of salivary calculi, and in cattle in connection with actinomycosis.

Anatomical Changes. Acute parotitis leads to swelling and intense reddening of the glandular tissue and to interstitial serous infiltration of the interlobular connective tissue (paro-

titis parenchymatosa); at other times it leads to the formation of small purulent foci between the lobules; these may subsequently become confluent and form larger abscesses (parotitis apostematosa s. abscedens). Chronic inflammation leads to an increase of the interlobular connective tissue, partial atrophy of the lobuli and induration of the whole gland (parotitis chronica indurativa).

Symptoms. **Acute inflammation** exhibits primarily a diffuse or on the contrary a more circumscribed swelling of the parotid region, which may extend into the neighborhood as a collateral edema. The edematous infiltration in the neighborhood leads, especially in cattle, to laryngeal stenosis and disturbances of deglutition. The gland itself is painful, and as in inflammation of the throat, the head is stretched, or in unilateral affections of the gland, held obliquely. The animals chew with care, and they do not ingest large morsels, because every depression of the lower jaw compresses the tender parotid gland. The secretion of saliva is usually increased (Müller), but it may also remain normal (Friedberger & Fröhner). Sometimes the disease is preceded by symptoms of stomatitis and pharyngitis, complicated with immobilization of the swollen tongue and intense swelling of the gums (Aruch).

Purulent inflammation always leads to inflammatory edema of the neighboring connective tissue, consequently the individual lobules cannot be distinguished; the swelling is usually of a high degree and continues down toward the neck and into the larynx. Later on the swollen tissues exhibit fluctuation in one place and pus finally breaks through in one or more places.

Every form of parotitis generally takes a favorable course. During the course of parenchymatous inflammation, the swelling goes down gradually and disappears rapidly after pus has been discharged spontaneously or evacuated surgically. The wound usually closes rapidly; frequently, however, a salivary fistula remains, which closes up finally under the proper treatment. Exceptionally the facial nerve of the same side may become paralyzed in consequence of compression by the swollen gland or of an extension of the inflammation along the nerve trunk.

Epizootic parotitis is accompanied by high fever; it may be unilateral or bilateral, and never results in suppuration. In cows it is sometimes accompanied by a mild catarrhal mastitis (Bissauge).

The chronic inflammation presents a firm, tough swelling of the parotid which is not painful. In making a diagnosis it is necessary to exclude swellings of a different nature (true tumors, etc.) which may occur in this region.

Diagnosis. Acute parotitis may be confounded with an inflammation of the neighboring connective tissue or of the re-

gional lymph glands. However, the swelling in these cases i more diffuse and does not reach up to the ear. The differential diagnosis may become very difficult since parotitis sometimes spreads to the neighboring connective tissue and may also be complicated by an affection of the lymph glands. Acute tuberculosis of lymphatic glands of the parotid region occurs in cattle and frequently also in hogs, and this fact must be considered in diagnosis. Pure cases of parotitis may be distinguished from pharyngitis by the absence of cough and nasal discharge and usually also of disturbances of deglutition; further, by the superficial character of the swelling in the parotid region. In catarrh of the guttural pouch there is a nasal discharge, while the parotid remains normal in size and consistency.

Treatment. To counteract the inflammatory swelling, Priessnitz' applications are indicated, possibly with carbolized instead of with ordinary cold water. Ointments favoring absorption, such as iodoform-, iodinevasogen-, camphor- or mercury-salves, are beneficial. Abscesses must be opened, the sooner the better. Chronic swellings may be treated by strong inunctions, injections of tincture of iodine, or of Lugol's solution; iodide of potash may be given internally. (Iodine treatment is particularly successful in actinomycosis.)

Inflammation of the submaxillary gland (Sialoadenitis submaxillaris), almost without exception, follows upon the penetration of foreign bodies, especially parts of food in straw feeding (Albrecht) into Wharton's duct. More rarely does it follow upon parotitis or stomatitis (authors' case).

The disease is most common among cattle, less among horses and least among dogs. One can feel the swollen, painful submaxillary gland upon the inner side of the angle of the maxilla. The margins of the meatus of Wharton's duct are often reddened and swollen; upon pressure on the duct, pus is frequently discharged. On the side and under the tongue a fluctuating or purely edematous and painful swelling may be recognized (Ranula inflammatoria) in contrast to a true ranula, which term is used to designate retention cysts on the floor of the mouth or upon the inferior surface of the tongue. More or less profuse salivation and disturbances of mastication likewise exist. This inflammation often leads to suppuration and more rarely to partial necrosis of the glandular tissue. Pus, usually fetid in character, breaks ordinarily into the buccal cavity, more rarely towards the outside; the disease then ends in recovery in one to two weeks.

The treatment is similar to that recommended for parotitis.

Angina Ludovici. This term is used in human medicine to designate an acute phlegmonous inflammation of the floor of the mouth, most frequently as a result of pus formation in the submaxillary salivary glands or the corresponding lymph gland. Roussel, Griffault and Marek have each observed a similar condition in dogs. Besides general symptoms and fever, a painful swelling appears in the submaxillary space accompanied by salivation, depressed appetite, difficult deglutition and pain on opening of the mouth. On raising the tongue there is found on the median line or to one side of the floor of the mouth a hard swelling which is sensitive to touch. It disappears in a few days either by way of absorption or by spontaneous discharge of the pus provided the pus has not previously been evacuated by a deep incision and thus a rapid absorption of the exudate brought about as in the case of Griffault.

Section III.

DISEASES OF THE PHARYNX.

1. Pharyngitis.

(*Rachenentzuendung, Halsentzuendung, Schlundkopfentzuendung, Halsbräune* [German]; *Angina.*)

As pharyngitis are designated all inflammations, which are partly catarrhal, partly deeper seated and penetrating even into the submucous tissue of the pharyngeal mucosa, including the soft palate and the tonsils. Such inflammations produce in man a constricting sensation of the pharynx during deglutition and they are therefore also called angina or synanche.

Occurrence. Pharyngitis is observed most frequently in horses and hogs, more rarely in cattle and still more rarely in sheep or carnivora. Among the first mentioned animals it occurs quite often enzootically, among the latter more rarely. Angina among chickens has been observed by Albrecht.

Etiology. Of external causes of acute pharyngitis, must be mentioned mechanical injuries by swallowing rough, pointed particles of food or other foreign bodies which penetrate the wall of the pharynx during deglutition or which injure the mucosa, and so form a portal of entrance for pyogenic bacteria. Hot feed or hot drinks, acrid, caustic materials may likewise produce an inflammation of the pharyngeal mucosa. This may also be produced by the inhalation of gases, hot air, or hot smoke during a conflagration. Sometimes gastrophilus larvæ produce pharyngitis either by direct mechanical irritation or by forming a portal of entrance for infecting bacteria. (Gastrophilus larvæ may also be an accidental finding in pharyngitis, as shown by the observations of Liehmann & Buffington.)

Colds sometimes play an important rôle. This is suggested by the frequency of the affection during the cold season when the animals are frequently exposed to the influence of sudden changes of temperature. Pharyngitis may appear, particularly in horses, a few days after exposure of heated animals to cold rain or sharp wind, or when cold air has come directly in contact with the pharynx. The ingestion of very cold, hoar-frosted

or frozen feed, or of very cold water may have a sim: ence. These influences are particularly potent in cattle when, during winter, they are kept in warm barns and are taken out to drink very cold water.

Infection undoubtedly often plays an important rôle. Even in those cases when the disease follows immediately upon a cold, the latter very probably only forms the predisposing cause. Suggestive for an infection as the cause is the fact of the enzootic appearance of the disease in certain localities with a tendency to progress from animal to animal. The bacteria concerned may be those which occur in the buccal cavity and pharynx of otherwise healthy animals (streptococci, bacillus necrophorus, bipolar bacteria) and which may only occasionally exert a morbid effect after the resistance of the mucosa has been lessened by noxious external influences. In the cases which evidently occur after cold, also after traumatic, thermic and chemical injuries, microorganisms probably stand in relation likewise, the external influences being only incidental causes.

The bacillus necrophorus deserves first consideration as it has repeatedly caused enzootics of angina in hogs (Johne & Meyfarth, Kitt, Schleg), especially in sucking pigs, two to four weeks old (Wyssmann), more particularly when the animals are kept under bad hygienic conditions, in cold, dark, badly ventilated barns, and if the mother animals are fed freely with flour. Schmiedhoffer found in the course of such enzootics paratyphoid-B bacilli as the cause and was able to produce with pure cultures a diphtheroid inflammation of the mucous membrane of the mouth and the pharynx of guinea-pigs and pigeons, and in the conjunctival sac and on the mucous membrane of the large intestines in rabbits. A similar croupous-diphtheritic pharyngitis was observed by Mayr, Strebel, Prietsch. Similar anginas have been observed in sheep (Roche-Lubin, Diem) and in dogs or cats (Robertson, Gray, Symes).

Young dogs sometimes develop an enzootic pharyngitis during the first weeks, which may pass into pyemia (Friedberger and Fröhner). In a case of croupous pharyngitis of a dog, Ball demonstrated streptococci which he believed to be the cause of the disease.

Cats are affected by an ordinary enzootic catarrhal pharyngitis without a known cause (Cadéac). (In Vincent's angina in man, a diphtheroid inflammation of the tonsils, fusiform bacilli mixed with spirillæ were found as previously mentioned.)

Secondary pharyngitis, as a part of the clinical picture of specific infectious diseases, frequently appears either sporadically or enzootically. Such diseases are strangles, influenza of the horse, buffalo plague, hemorrhagic septicemia of cattle and of swine, fowl cholera, anthrax, diphtheria, purpura hemorrhagica, variola, etc. Pharyngitis due to the bacillus suisepticus (septicemic angina) occasionally appears in enzootic form.

Inflammatory processes of neighboring tissues such as the nasal and buccal cavities, the larynx, the air sac, the esophagus, and of the bones of the face not infrequently extend to the mucosa of the pharynx.

A pharyngitis is sometimes produced on the ventral surface of the soft palate or on the lateral or dorsal wall of the pharynx by **Gastrophilus** larvæ being attached there, causing mechanical irritation or providing means for the entrance of micro-organisms. However, the finding of such larvæ may be only incidental in pharyngitis due to other causes, as the observation of Liehmann and Buffington seems to indicate.

Chronic pharyngitis is quite common. It is, however, of no great importance from a clinical standpoint, since it does not lead to important pathologic disturbances. It generally develops after an acute pharyngitis or after repeated, long-continued irritation.

Susceptibility. Horses and swine are most prone to develop disease of the pharynx. The marked difference in the predisposition of various groups of animals partially depends, perhaps, upon the fact that the tonsils of horses and swine possess several foramina coeca, those of other animals only single ones. Hence, it appears that the tonsils of horses and swine may more easily be invaded by pyogenic and other microorganisms than those of other animals. The existence of a broad ring formed by lymph nodes, tonsils, and diffuse cytoblast tissue, *in the pharyngeal mucosa, predisposes it in all animal species* to infectious inflammation produced first by bacteria carried to these tissues by leucocytes, which then pass over to the other portions of the mucous membrane.

Pathogenesis. The inflammatory process commences with hyperemia and often runs its entire course in the most superficial layers of the mucous membrane (catarrh), giving rise to copious exfoliation of epithelial cells, migration of leucocytes and production of a serous exudate which are mixed with the freely secreted mucus. In other cases a highly albuminous exudate forms which coagulates immediately, enclosing the migrated leucocytes and the dead epithelial cells (croup), or the tissue of the mucous membrane dies off in variable extent (diphtheroid) and after the necrotic tissue has been discharged, losses of substance remain (ulceration). In other cases the bacteria and their toxins are carried with the lymph stream into the interstitial spaces of the mucosa and of the submucous tissue, in consequence of which a sero-cellular or even a purulent infiltration may develop (phlegmonous inflammation) and give rise to abscess formation.

In purely superficial inflammation, microorganisms and their endotoxins are absorbed not at all or in very small quantities, in contrast to the other forms of inflammation in which large quantities of bacterial endotoxins enter the circulation and produce fever. Some of the microorganisms that are carried into the circulation by leucocytes, may escape lysis and localize, during the time of the pharyngitis, in other organs which are susceptible at the time, and cause inflammation in

them. Very often the microorganisms, with the products of their disintegration, are carried with the lymph stream into the mandibular and retropharyngeal lymph nodes and are arrested there, in consequence of which their invasion of the general circulation is prevented.

Functional disturbances in the pharynx occur in all cases, but are due, in purely catarrhal inflammation, exclusively to the increasing tenderness of the mucous membrane on swallowing; in severe inflammation, mechanical interference with deglutition is produced because the pharynx is narrowed by a bulging of the affected mucous membrane and at the same time the close approximation of the velum palatis to the dorsal wall of the pharynx during the act of swallowing becomes impossible. Further, the inflammatory infiltrated tissue in and between the muscles inhibits the contractions of the pharyngeal muscles and therefore the necessary raising in forward motion of the larynx. In consequence the transportation of the swallowed food into the stomach is more or less incomplete and, at the same time, the normally complete separation of pharynx from nasal cavity and larynx during swallowing, is incomplete. The epiglottis is raised up or pushed backward through the gelatinous infiltrated submucous connective tissue at its base, which interferes with respiration, as may also the not infrequent inflammatory swelling around the entrance to the larynx. The influence of pharyngitis upon respiration and swallowing varies considerably in different species of animals, probably because of anatomical differences, but principally because of the variable involvement of the submucous tissue.

Anatomical Changes. Catarrhal inflammation (pharyngitis catarrhalis) affects the mucosa only; this appears reddened diffusely or in spots, swollen, covered with a tenacious mucus; the lymph follicles and the mucous glands are swollen and the submucosa tissue may be in a condition of edematous infiltration.

In severe cases the highly swollen mucosa may become ulcerated and the submucous tissue then presents a thickened, gelatinous mass, containing streaky extravasations of blood, or purulent infiltrations (ph. purulenta s. phlegmonosa). Submucous abscesses may be formed in certain places, particularly in the retropharyngeal region (abscessus retro- or peripharyngeales). These then protrude into the pharyngeal cavity, encroach upon it, or may even obliterate it completely. After the opening of the abscess into the pharynx or towards the outside, there may exceptionally remain a pharyngeal fistula. After injury of the pharynx or after malignant infection, the abscess frequently contains fetid, ichorous masses, sometimes mixed with remnants of food. Retropharyngeal abscesses may also be formed in consequence of suppuration of the post-pharyngeal

lymphatic glands. Such abscesses are formed in the horse in strangles or influenza. In the dog, retropharyngeal abscess often appears simultaneously with purulent parotitis.

In some cases pseudomembranes form on the mucosa; they may consist of a deposit of exudate (ph. membranacea s. crouposa) or they may contain necrotic tissue (ph. diphtheritica).

The tonsils are always more or less swollen, their crypts filled with a thick purulent caseous, ill-smelling material. The soft palate is likewise swollen; the connective tissue between the bundles of muscle fibers is infiltrated edematously.

In pharyngitis in hogs due to the bacillus necrophorus the tonsils are much swollen, dirty grayish-yellow; the crypts are filled with dry caseous material, the lymph follicles in the neighborhood and in the mucosa of the pharynx in general are necrotic, the surfaces coated everywhere with masses of fibrin. The necrosis sometimes spreads to the surface of the tongue, while the submucous tissue of this organ and of the pharynx, and likewise the muscles become infiltrated edematously. The neighboring lymph glands show acute swelling. In pharyngitis due to paratyphoid infection, firmly attached lemon-yellow deposits equal to the size of a dime are observed on the root of the tongue, on the tonsils, on the lateral walls of the pharynx, on the epiglottis and even on the vocal cords and the lateral walls of the larynx. The other lesions are the same as in necrobacillosis of the pharynx.

In **chronic pharyngitis**, the mucosa is thickened, particularly on the posterior wall, and is bluish-red; the lymph follicles and mucous glands form nodules of the size of a lentil (ph. granulosa). Again, the mucous membrane of the pharynx may be studded with papilla-like granulations (Kech), while the muscles of the pharynx may be degenerated and show an increase of connective tissue (Trasbot). The tonsils of the horse and the dog may become swollen to such an extent as to form bluish-red cords equivalent to the thickness of a finger (Kitt, Marek). Liénaux and Liégeois described a special adenoid form of pharyngitis in young horses (Liégeois found it in 3 out of 400 horses) in which the mucous membrane of the anterior part of the pharynx was from 6 to 10 mm. thick and studded with lymphoid tissue, partly developed in the mucous membrane and partly projecting above the same as verrucose proliferations. The ailment was supposed to have been due to the usual causes of pharyngitis assisted by the favorable influences of a special diathesis and reminded one of the so-called adenoid growths in man.

Symptoms. In **acute pharyngitis** of horses and ruminants the ingestion of food occurs slowly and carefully and there is difficulty in deglutition. The food is masticated slowly and the head and neck are stretched out in swallowing. Sometimes food is expelled after repeated unsuccessful attempts at swallowing, to be again taken up later. Finally, the animals do not touch their food any more, and rather starve than be again and again exposed to the pain of swallowing. The ingestion of fluid, while easier, also causes pain, so that the patients may even refuse to drink, but only irrigate or wash

out their mouth in water that is held before them. The patients sometimes make swallowing movements, probably in consequence of the inflammatory irritation and of the masses of mucus which are collecting. Vomiting is brought about similarly, particularly in dogs and swine.

In a large number of cases, however, usually only in horses, ingested water is partly expelled through the nasal cavities (regurgitation). The cause of this symptom lies in the edematous infiltration which, in the severe inflammations, develops in the submucous connective tissue of the soft palate, the lower portion of the pharynx and between the muscles of the roof of the tongue. This interferes mechanically with deglutition and with the proper contraction of the muscles.

Nasal discharge occurs during the acute course of the disease, and the mucous secretions usually mixed with particles of food appear in large amounts at the anterior nares. A low position of the head in horses sometimes increases the nasal discharge. More or less salivation is likewise present. Increased tenderness of the pharynx is manifest in a stiff, stretched position of the head and, on moving, the animals hold the head and

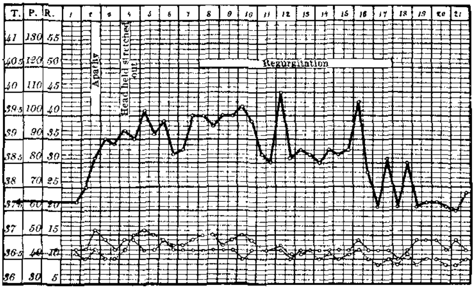


Fig. 4. Fever curve in a case of pharyngitis in a horse.

neck in this stiff position. They try to escape palpation and even a careful touching of the region of the larynx excites manifestations of pain and often leads to a convulsive cough, accompanied by a peculiar, snorting sound. The pharyngeal region frequently appears swollen, tender and hot. The swelling is particularly noticeable in hogs and the mucosa is purplish. The submaxillary and upper salivary glands are swollen, although they may not be palpable, in consequence of edematous infiltration. Sometimes swelling of the salivary glands is noticeable.

Direct examination of the pharynx shows increased redness of the ventral surface of the soft palate (velum palatis) and the fauces, in smaller ruminants also of the tonsils and the root of the tongue. The hand introduced into the pharynx meets with an increased resistance, it may feel the swollen fauces and the gelatinous infiltration of the epiglottic region at the base of the tongue, also remnants of food which are attached to the mucosa. *By artificial light with the rhino-laryngoscope, the* mucosa of the dorsal surface of the palate and the lateral walls of the nasopharynx are found reddened, bleeding easily and covered with mucopurulent deposits. If the root of the tongue is much swollen, the erect epiglottis and the raised palate may cover the larynx entirely in the mirror.

Cough is produced not only if food or water go the wrong way, but occurs spontaneously and is easily produced artificially. The cough takes place with open Rima glottidis, it sounds prolonged, painful, moist, and is accompanied by an after-cough that can be heard from a distance. Labored breathing, possibly with audible stenosis-sounds, occurs only in very severe cases.

Elevation of the temperature exists in the majority of cases. In pharyngitis following a cold or an infection, fever is usually present from the start and the latter may precede the other symptoms for one or two days (**Fig. 4**).

Pharyngitis from traumatic **or** chemical causes usually begins without an elevation of temperature, and fever only appears later after ulceration or pus formation has occurred. An elevation of temperature recurring during the later course of the disease points to a penetration of the process into the deeper tissues. In these cases the fever then generally persists until the shedding of the necrotic tissues or until the spontaneous or artificial opening of an abscess, when the temperature usually goes down within one to two days.

The urine, in insufficient nutrition assumes an acid reaction, even in herbivora, and it may be quite thin for days. In*dican is increased in ichorous necrosis of tissues. Meyer dem* onstrated an increase in the number of leucocytes of the blood in acute pharyngitis.

In the case of **hogs**, the animals are less lively at the onset of the disease, and hold their heads stiff during motion. The food is taken up more slowly; occasionally vomiting is noted which may be repeated in the further course of the disease. At the same time it is evident that swallowing is more difficult and proceeds with more or less bending of the head, especially if the food is more solid. In the frequent cases of phlegmonous, croupous or diphtheritic pharyngitis, the appetite is soon suppressed entirely and swallowing becomes impossible. In consequence regurgitation is not common and the severe disturbances of deglutition may be recognized only by the fact that

the quantity of water does not diminish when the animals attempt to drink.

It is also only in cases of phlegmonous and pseudomembranous pharyngitis that the larynx is swollen, the swelling increasing rapidly, being hot, painful and usually red-blue, in animals with unpigmented skin. This swelling may extend to neck and chest. The respiration rapidly becomes labored, rattling or whistling, at first only on motion or on crying, later even while at rest. The animals then sit like dogs, holding their mouths open so that the blue-red discoloration of the tongue is visible. The slightest disturbance increases the dyspnea to attacks of suffocation, with cyanosis of the unpigmented skin.

On direct examination a copious mucous deposit is seen on the reddened oral mucous membrane; the palate is reddened, also the root of the tongue, and particularly the tonsils. In case of extensive pharyngitis, if the examination be made without exposing the animals to the danger of suffocation, the parts are found much swollen, a purulent or pseudomembranous deposit covers the tonsils or other parts of the pharyngeal mucosa, and occasionally the oral mucous membrane.

Cough is present in almost all cases; it is rasping, painful, occasionally accompanied by vomiting.

In hogs, only severe pharyngitis is attended by fever which is, however, of no particular type.

In **carnivora** the clinical picture is ushered in by general disturbances like depression, discomfort, increase of temperature, if the disease arises from a primary infection. The act of swallowing is always difficult and painful, which can be recognized by the fact that morsels of food, or fluids, are transported to the stomach with evident exertion, with repeated bending and lowering of the head; the animals occasionally paw their heads with their forefeet, dogs cry out and let food drop from the mouth. The swallowing is rarely quite impossible, and then the animals take liquid food or water into the mouth, but the quantity before them does not diminish but is rather increased by admixture of foamy saliva. Empty swallowing is frequent, as is also vomiting. In many cases salivation is noted. A visible swelling of the pharynx is not seen often; it occurs especially after perforation of the pharyngeal wall by foreign bodies, or in case of phlegmonous and diphtheritic pharyngitis.

The region of the pharynx is very often sensitive on pressure, which also gives rise to retching or vomiting.

The animals strongly resist manual examination and show signs of pain when the mouth is opened. The tonsils are swollen, red or brown-red, sometimes hemorrhagic, elongated, at times so closely approximated that only a narrow slit remains between them. The pharyngeal mucosa is coated with mucous or mucopurulent secretion, especially on the palate; the fauces are livid, unevenly granulated, occasionally covered

th pseudomembranes. In traumatic pharyngitis red, striated excoriations may be visible on the velum and on the dorsal wall of the pharynx.

Cough is a usual symptom, dyspnea infrequent. In cats especially, symptoms of acute nasal catarrh and of conjunctivitis appear.

The symptoms of **chronic pharyngitis** are in nature similar to those described above; they are, however, milder in character. Deglutition is difficult but not impossible; the pharyngeal region is swollen moderately or not at all; it is not very tender; the ingestion of food is frequently interrupted by attacks of cough and the anterior nares discharge a tenacious mucoid or mucopurulent secretion.

In those cases of chronic pharyngitis in which symptoms are observed, inspection or palpation reveals enlargement of the tonsils or increased connective-tissue formation. In chronic adenoid pharyngitis Liénaux and Liégeois observed, besides symptoms of chronic pharyngitis, periodic inspiratory sounds due to stenosis even when the animal was at rest.

Course. Primary acute pharyngitis usually takes a favorable course, except in hogs and cats, where the disease usually leads to a fatal issue. The symptoms appear in rapid succession, so that they are fully developed in two to three days; they then remain at their height for an equal period of time and recede gradually, so that at the end of the first, or during the second week, complete recovery occurs.

The course is more protracted when ulceration or suppuration of the deeper tissues occurs. In these cases all symptoms persist for a longer time or even increase in intensity. The fever goes up, remains stationary or becomes remittent until the shedding of the necrotic tissue occurs or the abscess opens into the pharynx or into the outer world. After opening of the abscess and the discharge of pus, the condition of the patient rapidly ameliorates and recovery takes place. A pharyngeal fistula, which may have formed, generally closes only after a long period of time. Occasionally a thickening in the pharyngeal wall may be formed, which may permanently disturb deglutition and respiration, particularly during work (Wetzl). A catarrh of the guttural pouch may occur in horses; in hogs, a tough, not painful, swelling may be formed after the opening of a number of small subcutaneous abscesses, which permanently interferes with deglutition and respiration. Paralysis of the pharynx may also occur, but may again disappear after a prolonged period.

A fatal issue is generally due to complications. Edema of the larynx, in the absence of proper aid, may lead to suffocation. *An occurrence, which is not infrequent in horses, is as-*

piration pneumonia due to swallowing or inspiration of pus of an abscess breaking into the pharynx. Inflammation may also spread into the loose connective tissue in the mediastinum, and then a pleuritis or pericarditis closes the chapter (Cadéac). In phlegmonous pharyngitis septic material may be absorbed from ulcerations and lead to general sepsis or a fatal hemorrhage may occur at the base of an open ulcer (Wakefield, De Jong, Monod).

Diagnosis. The clinical picture of pharyngitis is, as a rule, quite characteristic, so that diagnosis offers no difficulties. Foreign bodies which have become wedged in the pharynx, generally in carnivora and cattle (Fiebiger has seen two such cases in horses), can best be detected by inspection and internal manual palpation. (In the case described by Sustmann a hatpin was found in a swelling in the region of the eye. It had penetrated the wall of the pharynx and intervening tissue.) Tumors of the pharynx are usually not painful, the signs of an acute affection are missing and internal palpation reveals the tumor. In paralysis of the pharynx there is no tenderness and the peculiar position of the head is absent. Diseases of the central nervous system will lead to other paralysis besides that of the pharynx; and in cases of meningitis there are mental disturbances. In obstruction of the esophagus no changes are found in the pharynx and regurgitation during the act of deglutition occurs later; a sound will find the obstruction in the esophagus. Parotitis does, generally, not lead to regurgitation, and is not accompanied by nasal discharge. If pressure is made on the lower wall of the pharynx (base of the tongue), pain is not elicited. Catarrh of the guttural pouch generally causes a unilateral swelling in the upper portion of the parotid region; pressure on the latter usually causes nasal discharge; pressure on the lower pharyngeal wall does not elicit pain. Since pharyngitis may be a part of the picture of a general infectious disease, one must, in the beginning, think of this possibility. In horses one must think of strangles; in cattle and swine, of hemorrhagic septicemia, hog cholera, or anthrax.

The determination of the nature of pharyngitis is usually very difficult, except in smaller animals where inspection permits a good survey of the anatomical changes. It may be said, in general, that high fever persisting during the course of the disease, intense and increasing swellings of the pharynx, speak for a deeply penetrating inflammation. Gangrene of the mucosa, phlegmonous or diphtheritic inflammations, aside from their local manifestations, are characterized by a fetid smell of the saliva and the expired air. Coughed up masses of fibrin point to croupous pharyngitis. In proper cases one may use in horses the rhinolaryngoscope of Polansky & Schindelka, which occasionally makes it possible to recognize swellings of

the mucosa, hemorrhages, small abscesses, etc. In difficult respiration this instrument cannot be used.

Prognosis. The prognosis of primary uncomplicated pharyngitis is favorable; however, it becomes the more unfavorable the more the symptoms point to phlegmonous, diphtheritic or purulent inflammation. Even affections which are mild at the start, may in their further course assume a malignant character, particularly in horses, where aspiration may easily occur; hence a guarded prognosis is advisable during the early stages.

Treatment. Considering the increasing tenderness of the mucosa and the difficulties in deglutition, the food must be soft, mushy or fluid in consistency. In herbivora, the best nourishment is green feed, well wetted hay, flour or bran mashes thin in consistency, while hogs and carnivora are best fed with milk. Horses suffering from intense difficulty in deglutition should at first not be fed even with entirely fluid feed and water; thirst can be alleviated by repeated injections of lukewarm water into the rectum. The patient should fast from one to two days and artificial feeding may then be instituted (see page 43). This is best accomplished by the introduction of liquid food into the stomach through the stomach tube. Most patients permit the introduction of this instrument without any difficulty. If stenosis of the larynx exists at the same time, a preliminary tracheotomy must be performed. The authors have in this manner fed many horses for days and even for weeks and have thus prevented inanition and aspiration pneumonia. Cribs and utensils soiled with pathologic secretions must be cleansed at short intervals.

The local treatment consists in the application of astringent and disinfectant solutions, such as 2% nitrate of silver, 3 parts of tincture of iodine with 25 parts of glycerin, 10% alum-glycerin; peroxide of hydrogen, 15% creolin-vasogen. These medicated solutions are used to paint the pharyngeal mucosa; however this procedure can be employed only in short headed animals. If the animals so treated show too much excitement, it is better to desist from this form of treatment, likewise in larger animals when the difficulty in deglutition is not too great; in such cases disinfectants may simply be added to the drinking water, such as chlorate of potash (horses 40-50 gm. for each bucket of water, dogs a 2% solution in tablespoon doses), or salicylate of sodium (80-100 gm. or a 0.5% solution). Inhalations of the vapors of pure water, or of a 2% carbolic acid solution, or a finely divided spray of astringent or disinfectant solutions (1-2% carbolic acid, creolin, alum, or tannic acid, possibly corrosive sublimate 1:1000) may also be used. It appears best to introduce these solutions with a Frick or a Bayer-Kieselbach spraying apparatus. In horses which are

not particularly restless these astringent and disinfectant drugs may also be applied in the form of ointments. This can be done with the aid of a long, metallic sound. Its dull end is wrapped in cotton, saturated with the ointment (according to Bringard eucalyptol and vaselin 1:15); the sound is introduced below the inferior turbinate into the pharynx, and the ointment is rubbed over the dorsal surface of the soft palate and over the mucosa of the naso-pharynx. Intrapharyngeal injections from the trachea as recommended by Dieckerhoff are not always devoid of danger; the same is true of the intranasal injection of fluids through a hard rubber tube or a urethral catheter. Hungarian veterinarians (Sulyok, Heim, Axmann, Schmiedhoffer) have obtained surprisingly favorable curative results in pseudomembranous pharyngitis in pigs by the injection of diphtheria antitoxin (20 cc.). According to Schmiedhoffer's experiments equally good results can be obtained with normal horse serum, which, moreover, can be easily procured on the premises when needed. External applications in the region of the pharynx are always beneficial. In the earlier stages cold applications are indicated, later on Priessnitz' applications, with disinfectant solutions (three per cent carbolic acid, 1 to 1000 corrosive sublimate solution, spirits of camphor) are advisable in all cases. Warm applications (with oat, barley or linseed meal, etc.) may be used in case of continued swelling to hasten the ripening of the abscess. Ointments favoring resorption (gray mercurial ointment, camphor or iodoform ointment 1:10) may likewise be useful. If the swelling shows no tendency to go down, inunctions may sometimes give relief in combination with Priessnitz' applications (oil of mustard in 6% to 8% alcoholic solution, red biniodide of mercury ointment 1:4).

Abscesses should, if possible, be opened early; it is sometimes possible in cattle to open an abscess, previously located in the pharynx by palpation, with a knife introduced into the mouth (Macgillivray, Cunningham). A pharyngeal fistula which has eventually formed may be made to heal within a few weeks by repeated disinfection. Tracheotomy must not be delayed in the presence of intense dyspnea or edema of the larynx. The use of internal medicines usually appears superfluous and may even be dangerous. Should it however become necessary, such medicines should be used by rectal or by subcutaneous injections.

Chronic pharyngitis in small animals may be treated with local applications of tincture of iodine (Tinct. Iodi., Tinct. Gallarum āā), chloride of iron (1:6), iodine or tannic-acid-glycerine, insufflations of boracic acid or tannic acid with sugar (āā). In large animals treatment must be limited to irritant inunctions and Priessnitz' application.

Artificial Feeding of Sick Animals. The simplest and most practicable method of feeding consists of introducing into the stomach nutriments in the simplest form. This is best accomplished by drenching the animals with these nutriments in liquid form by using a thick-walled bottle or by attaching a rubber tube to a funnel or a fountain syringe. Small animals may be fed by means of a spoon. Flour and grain gruels are especially well suited for herbivorous animals, while milk, bouillon or soups made from flour to which a little glucose and a few eggs have been added are best suited for carnivorous animals. In cases of difficult deglutition or if coughing is provoked it is best to refrain from this method and to introduce the food directly into the stomach by means of a stomach tube. Artificial feeding *per rectum should be resorted to in all cases* of sensitiveness of the pharynx, obstruction of the esophagus, gastritis or enteritis, or when, because of excitement of the animal or other condition, the passage of the stomach tube is impossible or its use is contraindicated. Fifteen to thirty minutes before the rectal feeding the feces which have accumulated in the rectum should be removed with the hands or by means of an enema. Liquid nutriments may then be introduced into the rectum by means of a fountain syringe, those of a thicker consistency with some other suitable syringe under light pressure. These substances should be introduced at body temperature and as high into the rectum as possible. *One should seek to prevent the expulsion of* the enemas by pressure on the tail or by the addition of opium. It seems advisable to add to the enemas 1 to 2% sodium chloride, which, by promoting antiperistalsis, will carry the liquid to the anterior portion of the intestines and also facilitate absorption. When enemas are used daily the rectum should be thoroughly flushed once daily in order to remove any accumulated and decomposing substances.

Small dogs and cats should receive 10 to 40 cc.; large dogs, 100 to 200 cc.; small ruminants, colts and calves, 200 to 400 cc., and adult horses and cattle, 2 to 3 liters of such food material, three to four times daily (Jakob).

Regarding the composition of the food enemas, one must keep in mind that the mucous membrane of the large intestines can only convert starch and cane sugar into glucose but at the same time possess quite a capacity to absorb water, salts, glucose, peptones, albuminates and other proteins as well as small quantities of emulsified fat. For dogs such food enemas are best prepared from milk, *starch and glucose or from raw eggs (Ornstein).* They should not contain more than 2% glucose nor more than 6 to 10% starch. The starch is first placed in a previously heated vessel and by the addition of boiling water is made into a uniform thin paste. The glucose is dissolved in warm water and added to this paste while still hot. After the mixture has cooled to body temperature, add 2 to 3 eggs which have previously been beaten with a tablespoonful of water and a pinch of salt.

The food enemas recommended by Leube for the artificial feeding of man may also be used. Such are peptone-milk enemas (250 gm. milk, 60 gm. peptone); egg-milk enemas (250 gm. milk); sugar enemas (60 gm. glucose, 250 gm. milk); pancreas enemas (50 to 100 gm. pancreas, 150 to 300 gm. meat, 30 to 45 gm. fat). In its simplest form the meat-pancreas enema may be prepared by mixing finely minced beef with finely minced, fat-free pancreas from the hog (3 to 1), to which mixture a little fat (for example, 30 to 40 gm. cod-liver oil which has previously been shaken with a few tablespoonsful of a 0.3% soda solution) and 6 to 10% starch, or, better, glucose not to exceed 2%, is added. In place of fresh pancreas pancreatin may be used, while for pet animals the fresh meat may be replaced by artificial peptone preparations (peptone, meat meal, roborat, tropon, kalodal, etc.).

Artificial feeding by subcutaneous injection may also be considered, especially if a diseased condition of the large intestines make the use of food enemas inadvisable, or when such enemas are immediately expelled. According to Ornstein, 150 cc. of blood serum with an addition of 10 to 20% glucose are entirely absorbed and assimilated. This amount will save the catabolism of 22% protein of the body. The most practicable method of introducing these fluid nutriments is to inject them by means of an infusion apparatus with the application of a low pressure. The blood serum should first be boiled and injected in a sterile condition and at body temperature. Blood serum foreign to the species should only be used after it has been stored for several days or under special precautions (see serum sickness) in order to prevent anaphylactic symptoms. Serum of the same species is better for this purpose, but even sera of a different species will lose much of their toxic character by heating for some time at 55 to 68° C. Kalodal (phosphorus protein compound) is considered nontoxic when given sub-

cutaneously. It is given to dogs in a 1% solution. This solution is prepared by dissolving 5 gm. in 50 cc. distilled water, sterilizing the same by boiling and then diluting with 500 cc. sterile physiological salt solution. Since only traces of fat are absorbed, its use does not seem advisable unless fat taken from the same species can be used. Fats (oil, butter, lard) may be given to large animals in doses of 50 to 70 gm., to small animals in doses of 10 to 15 gm., injected subcutaneously at several places, repeating the dose four times a day. The dose of a mixture of blood serum and 10 to 20% glucose or of glucose alone (10% glucose in physiological salt solution) for dogs may be as high as 200 gm., while large animals may be injected with a liter. Benesch never obtained good results by artificial feeding per rectum, but secured very reliable results in dogs from subcutaneous injections of sterile olive oil at intervals of 4 to 5 hours and using 10 to 15 cc. at each injection (up to 60 cc. per day).

For intravenous feeding glucose may be used in an isotonic solution (4%). When possible blood serum from the same species should be mixed with the glucose in order to provide the necessary protein.

Stomachal artificial feeding is undoubtedly the most important, since it more nearly approaches physiological conditions and permits the introduction of a large quantity of energy-producing material. In this way even large animals may be kept alive for weeks. On the other hand rectal feeding or parenteral artificial feeding can only reduce the catabolism of substances stored in the body but does not suffice to keep alive for a long period animals that do not get other foods. Its long-continued application also meets with technical difficulties in practice. By the daily use of food enemas a catarrhal condition is usually produced in the mucous membrane of the rectum, even when easily spoiled substances, such as eggs, are used for a period not longer than three days and the concentration of glucose kept very low. In subcutaneous feeding, on the other hand, abscess formation or necrosis of the skin may take place at the point of injection, even when the greatest precautions are taken, while intravenous injection can not be repeated often even in large animals.

2. Animal Parasites in the Pharynx.

Gastrophilus larvæ (**Gastrophilus hemorrhoidalis** and **G. equi**) in horses are sometimes found attached to the posterior and lateral walls of the pharynx, but especially to the ventral surface of the velum palatis and to the posterior pillar of the soft palate. They occasionally localize in large numbers (in the case of Limdenau up to 50, in one case of Schlegel more than 31 and in one of Sittler 46) and produce severe pharyngitis and proliferation of tissue, or, without such results, cause severe disturbance in deglutition, sometimes even total inability to swallow, coughing with spasms, suffocation, difficult breathing, and sometimes foreign-body pneumonia. The local disturbance can be quickly remedied by removing the attached larvæ from the ventral surface of the velum palatis with the use of a speculum and painting the point of attachment with iodin, and then applying to the mucous membrane of the pharynx an iodin-glycerin solution by means of a cotton swab attached to the end of a rod and saturated with this solution.

The **Rhinoestrus (Oestrus) purpureus** (Cephalomyia purpurea) occasionally lives in horses in the nasal cavities and in the accessory cavities, also in the pharynx. It causes pharyngeal catarrh, cough, or even dyspnea with audible sounds of stenosis after advancing to the entrance of the larynx. Similar symptoms are produced occasionally by the Cephalomyia maculata, in buffaloes, camels and dromedaries.

Far more dangerous is the **Cephenomyia trompe,** the nasal fly of reindeer. According to Bergman's observations the adult fly frequents the vicinity of reindeer herds in the summer months not later than the beginning of September. The female fly pursues the reindeer by hovering in the air under the neck, and at a favorable opportunity approaches a nostril and deposits, or possibly even injects, therein a drop laden with larvæ. At the approach of the adult fly the reindeer frequently stand with lowered head, often hide the mouth and nose in moss, and

after deposition of the larvæ throw the head from side to side, suddenly snort repeatedly, and anxiously rub the nose on the ground or on the forelegs. The nasal bot-fly of the reindeer as a rule succeeds in depositing its larvæ in almost every reindeer. The larvæ then preferably attach themselves in the sac-like depressions of the caudal pharynx and frequently produce severe pharyngitis, rhinitis and laryngitis or even foreign-body pneumonia, also bursting of the sac-like depressions of the pharynx, causing a progressive phlegmonous inflammation in the region of the trachea. In case the invasion is severe calves and feeble animals perish from exhaustion. The losses may amount to 1% to 2% of the herd (Pawlowsky, Kerzelli). Cattle are also frequently pursued by the nasal bot-fly of the reindeer. In them the deposited larvæ, by irrigation of the nasal mucosa, induce sneezing for hours with discharge of thin mucus and licking of the nose, but do not undergo further development. Similar affections may be caused in reindeer and stags by œstrus larvæ.

The **Oestrus larvae** are removed from the pharynx by means of a stick covered with oiled linen, also with eucalyptus ointment (1:15) which is applied by means of a cotton pad attached to the stick.

Hungarian flies (Simulia columbacsensis), when attacking cattle in larger numbers, are liable to wander through the mouth and nose into the pharynx, where they cause a profound inflammation leading to suffocation.

Horse-leeches (Hæmopis sanguisuga), in southern countries, sometimes get with the drinking water into the pharynx of horses and mules; more rarely of cattle; they suck the blood from the mucosa and produce inflammation. Their presence is indicated by a considerable hemorrhage from the mouth or nose. If present in larger numbers, they may lead to profound anemia or even fatal hemorrhage (Baise saw in two cases 185 and 192 leeches).

Prophylactic measures in southern countries consist in filtration of the drinking water. The treatment of the affected animal consists in irrigation with solutions containing common salt, vinegar or ammonia or in the inhalation of turpentine vapors.

Tokishige found filiform worms in the pharynx of a horse dead from pharyngitis, which he thought to be **Dispharagus reticulatus.**

3. Tumors of the Pharynx. Tumors intra-et peripharyngeales.

Occurrence. Tumors of the pharynx and its neighborhood are rare in domestic animals. Least frequently seen are such true neoplasms as papilloma, sarcoma, carcinoma, lipoma, also retention cysts; connective tissue hypertrophy of the mucosa of the pharynx is seen after an acute pharyngitis (Wetzl). Actinomycomata or tuberculous growths are seen more commonly in cattle. Old abscesses in the retropharyngeal region in horses and cattle have a similar significance clinically.

According to Zimmer's statistics seventy-three cases of tumors of the pharynx in cattle were divided as follows: Fifty-four cases of actinomycosis, five cases of tuberculous growths, seven cases of dermoid cysts, four cases of fibroma, two cases of colloid cysts, one case of melanoma. Out of 4,708 cases of profound tuberculosis Rasmussen found retropharyngeal lymph glands in a condition of tuberculous degeneration in 3,245 cases (68.93%); the gland below the parotid in 228 cases (4.84%); and the tonsils only in fifty-two cases (1.1%).

As has been shown by the fundamental work of Mörkeberg, actinomycosis usually develops in the upper, posterior parts, more rarely in the lateral walls of the pharynx, that is either in its mucosa or in the neighboring tissues. These actinomycotic masses then show a tendency to project polyplike into the pharynx. Tuberculous tumors, on the contrary, arise outside of the pharynx and only make its wall protrude in a tumorlike manner. The root of the tongue is usually the seat of retention cysts, also the anterior surface of the epiglottis or a place between the latter and the root of the tongue. True tumors usually arise in the upper posterior portion of the pharynx.

Intrapharyngeal tumors usually become pediculated in consequence of traction made in deglutition.

Symptoms. The sessile tumors produce increasing difficulty in swallowing, but without pain; later dyspnea and rattling, whistling and snorting noises are heard, when the tumor encroaches upon the posterior nares and the larynx. Difficulty in deglutition and respiration is increased when the head is bent forward or sideways. Stenosis of the posterior nares causes labial breathing in all animals (bulging of the cheeks during expiration) which disappears when the mouth is opened. The difficulties in swallowing and the respiratory disturbances are increased by turning the head inward and sideways.

Tumors which are not very voluminous cause disturbances in swallowing or in respiration only at intervals if, during the act of swallowing and on deep inspiration or when the head is held in a certain position, they are thrown into the first portion of the esophagus, into the posterior nares or into the larynx. An attack of coughing or vigorous shaking of the head removes the tumors and the disturbances cease. Such an attack may cause death by suffocation. As the tumor increases in size, the attacks become more frequent, and finally the difficulties in swallowing and breathing become permanent.

Smaller, pediculated tumors cause difficulty in deglutition or respiration as well as rattling sounds only occasionally, namely, when they become displaced into the pharynx, larynx or towards the choanæ, in deglutition, strong inspiration and in certain positions of the head, until they are again expelled by a strong expiration or a movement of the head. It may, however, occur occasionally that such an attack brings abont suffocation of the animal. As the tumor grows the attacks become more frequent and lead finally to permanent difficulty in deglutition and respiration.

Discharges from the nose are only observed in part of the cases, such discharges are sometimes fetid and mixed with fragments of tissues. Epistaxis occasionally occurs; dogs sometimes vomit and the tumor may then be visible in the buccal cavity.

With the exception of those on the posterior surface of the soft palate, tumors of the pharynx in smaller animals can be seen on inspection; in horses, however, only with the aid

of the rhinolaryngoscope. With the hand introduced into the pharynx even those tumors can be palpated which arise in the neighborhood of the posterior nares.

Peripharyngeal tumors when present produce visible changes in the pharynx.

Swelling of the submaxillary, also occasionally of the peripharyngeal lymphatic glands, occurs in malignant tumors and tuberculosis of the pharynx.

Diagnosis. The described disturbances of deglutition and respiration, if developing slowly, strongly suggest the presence of pharyngeal tumors; a definite diagnosis can however be made only after local inspection. When the examination of the pharynx is negative, tumors of the larynx may be discovered by laryngoscopy and it may perhaps be necessary to make an exploratory laryngotomy.—Tumors of the larynx may also be distinguished by the fact that compression of the larynx increases the dyspnea; this is not the case in tumors of the pharynx. However, exploratory tracheotomy may be necessary to settle the point.—Nasal stenosis can easily be excluded by a local inspection.—Retro- and peripharyngeal abscesses furnish a similar clinical picture during a protracted course, but local examination and the history of the case permit a correct differential diagnosis. In making the latter the following other conditions have to be considered: Chronic disease of the pharynx, and of the guttural pouch, spasm of the glottis, stenosis of the bronchi, and in the case of dogs cardiac dyspnea in uncompensated valvular disease of the heart, which likewise leads to attacks of rattling respiration.

The determination of the nature of the tumor requires a careful examination of the pharynx and its neighborhood.

Prognosis. This depends upon the seat and the nature of the tumor. If the latter can be removed, the animal can be saved; if its removal is impossible the animals die from suffocation, pneumonia or marasmus. Even tuberculous tumors may be removed and complete recovery may follow the operation if the process has been purely local. Tuberculous and actinomycotic tumors sometimes become smaller and shrink after the evacuation of their contents.

Treatment. Pediculated tumors and those not having a broad base may be crushed or cut off after ligation of the pedicle with instruments iutroduced through the mouth. A preliminary tracheotomy is however usually necessary. Iodide of potash often proves of no value in the treatment of actinomycotic tumors of the pharynx. (Details about the operative treatment of pharyngeal tumors may be fouud in the publicatious of Mörkeberg.)

SECTION IV.

DISEASES OF THE ESOPHAGUS.

1. Inflammation of the Esophagus. Oesophagitis.

(*Entzüendung des Schlundes* [German].)

Etiology. Irritating, caustic substances or rough, pointed bodies, which are ingested with food or water, or improperly selected medicines, may get into the gullet and cause catarrh or a rather penetrating inflammation of the esophagus. Their injurious influence here becomes more rarely manifest than in the buccal cavity or pharynx, because the esophageal mucosa is less sensitive and better protected by a thicker epithelial covering, and also because irritating matters very rapidly pass through the gullet. These morbid conditions are met with after the ingestion of very hot distillers' slop, after the administration of ammonia, tartar emetic, after poisoning with acids or alkalies or after greedy swallowing of rough fodder, fragments of bones mixed with meat, foreign bodies which become wedged in the gullet, or after injury by the esophageal sound. Finally, there are traumatic influences acting from without which may become the cause of the inflammatory process. Cannon observed in a horse suffering from gastrophiliasis of the pharynx an inflammation produced by **Gastrophilus** larvæ **(G. pecorum)** and in one place perforation of the esophagus in the vicinity of which 70 larvæ, partly in groups, had deeply penetrated the muscles. Schlegel found a phlegmonous and purulent inflammation of the esophagus in 19 cattle at pasture in the time between the months of January and March.

The disease may be secondary to inflammatory processes of the pharynx or the stomach, especially if these are due to a general infection (foot-and-mouth disease, variola, diphtheria, rinderpest, etc.).

Anatomical Changes. In superficial inflammations the epithelial covering is missing either in irregular patches or sometimes to a larger extent, or even along the whole length of the esophagus (Berthéol). The mucosa appears dark red on the uncovered portions, occasionally hemorrhages are seen here; in the other portions the epithelial layers are loosened

and can be removed easily; the submucous connective tissue exhibits a more or less intense edematous infiltration. In severe cases the wall of the esophagus is thickened, swollen and the subcutaneous and intramuscular tissue is the seat of a gelatinous or even purulent infiltration. Similarly, however, more severely affected, especially posteriorly, appeared the esophagus in the cases of Schlegel, involving **Hypoderma** larvæ infestation, only there were found in the tumefied submucosa in the muscular layer, and especially in the periesophageal connective tissues, bandlike burrows or necrotic purulent foci with numerous translucent **Hypoderma** larvæ 1.2 cm. long and 1.5 cm. thick. Cattle have developed croupous inflammations after the instillation of spirits of ammonia (Lemaire).

Chronic catarrh leads to a marked thickening of the epithelial layers, occasionally also to papilliferous proliferations.

Symptoms. A very superficial catarrh of the esophagus usually escapes notice. In somewhat more intense cases the food is swallowed with a great deal of effort. The animals move the head restlessly from side to side in swallowing, stretching and bending the neck, horses paw with their front feet and their faces express anxiety. Sometimes one may observe how a morsel becomes wedged in the esophagus, occasionally directly below the pharynx. Such morsels then become dislodged toward the pharynx by antiperistalsis and are expelled through the mouth or nose. Esophagitis in cattle due to **Hypoderma** larvæ manifests itself in difficult deglutition, disturbed rumination and loss of flesh. Deeply penetrating inflammations lead to a mucous secretion or to a bloody discharge from the mouth or nose independently of the ingestion of food. In such cases food is refused. Tubular croupous masses are sometimes expelled in croupous inflammation. Increased tenderness of the esophagus is shown by the animals when pressure is made over the left jugular depression. One may sometimes see here spontaneous undulating motions followed by the expulsion of mucus or food from the mouth. In more intense inflammation of the esophagus, particularly in carnivora, vomiting occurs. Difficulty in deglutition greatly interferes with the nutrition of the animals and the inflammatory process may cause an elevation of temperature.

Course. Simple catarrh ends in recovery after one to two weeks, while deeply penetrating inflammations lead to cicatricial contractions of the esophagus or to a purulent infiltration in the surrounding connective tissue. The infiltration spreads along the large vessels towards the thoracic cavity and leads to the formation of abscesses. Exceptionally a perforation of the esophagus occurs. All these complications are indicated by an inflammatory swelling in the region of the left jugular

vein. If perforation occurs in the thoracic portion of the esophagus pleurisy follows.

Treatment. One should attempt to ameliorate the inflammatory process by the administration of small pieces of ice, by cold water, cold milk, mucilaginous or astringent fluids (½ to 1% tannic acid in linseed infusion). Cold applications on the region of the esophagus should also be made. If the inflammation is due to corrosive poisons, antidotes like weak acids or alkalies in mucilaginous milk should be administered. When the pain is intense narcotics are indicated (morphine subcutaneously, chloral hydrate, tincture of opium in the drinking water). If the animals still persist in refusing to take food, artificial feeding per rectum becomes necessary.

2. Spasm of the Esophagus. Oesophagismus.

(*Krampf des Schlundes* [German].)

Spasm of the esophagus consists in a morbid contraction of its muscles, which occurs in paroxysms, without organic disease or obstruction. These latter conditions also lead to spasm of the muscularis of the esophagus. Esophagism, as a disease per se, is rare among domestic animals.

Etiology. Primary disease of this kind is very rare, and it then occurs upon a neuropathologic basis. It has been observed without any special cause only a few times in nervous horses of quick temper. Such animals may have an attack upon drinking cold water or upon pressure upon the esophagus. Nothing definite is known whether in these cases disease of the pneumogastric nerve or transitory malposition of the esophagus may play a rôle.

Horses sometimes exhibit esophagism after the injection of morphine or after chloral hydrate or chloroform narcosis (Fröhner, Vennebrolm). In a case of this kind Plósz could demonstrate the obstruction of the esophagus by a morsel of food. A similar morbid condition occurs very rarely in adult cattle (Ries) and somewhat more frequently in calves (Troussier).

Secondary spasm of the esophagus occurs in the course of other esophageal diseases, especially in inflammation and obstruction, also in tetanus and in rabies. It is not certain whether lesions of the vagus may be of influence in many cases.

Symptoms. When the spasm begins, the animal suddenly becomes very restless and betrays great anxiety; if it happens to be feeding when the spasm comes on, the ingestion of food ceases, yet empty mastication continues, and a foamy saliva collects in the mouth. The animal stretches its feet out and tries

to swallow the saliva while the head is strongly stretched or bent. At the same time, the undulatory motions of the esophagus become visible in the left jugular region. However, swallowing of the saliva can take place only incompletely, and it is expelled through the mouth and nose by antiperistaltic movements. If the spasm occurs during the ingestion of food, the saliva is at first mixed with food particles, later on it becomes pure and does not give an acid reaction like expelled gastric contents. Along the left jugular depression the cordlike gullet can be felt distinctly, pressure upon it excites pain. Sometimes a moderate enlargement, filled with air, saliva and mucus, is formed above the spasmodically closed section (Friedberger).

The attack may last from a few minutes to several hours; it may or may not recur. In a case reported by Cadéac, the sick horse had several such attacks every week for a year and a half, and these could be produced at will by feeding short cut dry feed or by the administration of cold water. Roy, on the other hand, saw five attacks in five years; they always lasted five hours and were followed by respiratory difficulties lasting for twenty-four hours. The disease always ends in recovery.

Diagnosis. On account of the disturbances of deglutition and the subsequent vomiting, esophagism may be confounded with inflammation, stenosis or obstruction of the esophagus. However, the sudden appearance, the likewise rapid disappearance of the attack, the perfectly normal condition of the animal between the attacks furnish enough data to distinguish this affection from stenosis or inflammation. Primary esophagism may be distinguished from mild cases of obstruction of the gullet in the thoracic portion, which disappear spontaneously after a short time, by the use of the sound (perhaps after the preliminary application of narcotics). If no impediment is found and if the attacks still persist, the diagnosis is, of course, primary esophagism. It is also possible that during very greedy feeding, a morsel may become lodged in the caudal portion of the esophagus, this subsequently may pass on, yet the described symptoms may persist a while, and a veterinarian not knowing the history of the case may think of primary spasm of the esophagus (Cagny, Johne). Indeed, esophagism is, as a rule, due to a temporary obstruction or to a superficial injury.

Treatment. The disturbed condition of the nerves calls for the use of narcotics, such as morphine subcutaneously (0.4-0.6 gm. for horses; 0.01-0.1 gm. for smaller animals); chloral hydrate per rectum (25-50 gm. for large animals; 0.05-0.5 gm. for smaller animals); during the intervals between the attacks bromide of potash internally (for horses, in daily doses of 20 gm.). This treatment is usually successful.

3. Paralysis of the Esophagus. Paralysis oesophagi.

(*Laehmung des Schlundes* [German].)

Etiology. Paralysis of the esophagus occurs usually together with paralysis of the pharynx, and is due to the same causes as the latter. There have, however, been described a few cases of primary paralysis of the esophagus in horses, the nature of which has not been cleared up. Moeller saw this affection a few times after resection of the arytenoid cartilages. He found marked proliferation of connective tissue around the larynx in one case and thinks it may be possible that in other cases likewise inflammatory swelling in the first portion of the esophagus might have been the cause of the disturbed deglutition. In a case of Brissot, a fall upon the left side, and in a case of Graf, a kick on the neck were the causes of paralysis of the esophagus. (In these two cases a traumatic lesion of the esophageal wall itself cannot be entirely excluded, also in the case of a horse of the Prussian army.) In this case a gelatinous bloody infiltration was found behind a portion of the gullet filled with particles of food. Theiler observed several cases of paralysis of the esophagus in horses, but seldom in mules, following the dikkop form of horse sickness in artificial as well as natural infection. The cause must probably be ascribed to toxins of the infecting organism acting upon the nerves of the esophagus, but also to pressure upon these nerves produced by the gelatinous infiltrated connective tissues in the region of the pharynx. Puschmann and Schneider were unable to discover a cause in their cases.

Occasionally histologic changes may be discovered in the pneumogastric or recurrent nerves.

Symptoms. In primary paralysis of the esophagus the food is always masticated without trouble; but masses of fodder accumulate in the gullet. In this manner the esophagus forms a thick, firm or more soft, non-painful, cylindrical prominent mass in the left jugular depression. Deglutition becomes impossible and regurgitation occurs. These disturbances appear suddenly.

Disturbances in deglutition coming on after arytenectomy may disappear after a few weeks. In the above mentioned case of Brissot recovery occurred in two days, but the affection may lead to death in consequence of impeded nutrition or of foreign body aspiration pneumonia.

Diagnosis. A diagnosis of paralysis of the esophagus can be made after the exclusion of paralysis of the pharynx, obstruction of the esophagus, stenosis or dilation of the esophagus, contusion of the esophagus.

Treatment. Attempts must be made to push the accumulated, wedged-in masses of food down with the esophageal sound. The animals should then receive juicy or liquid feed only. The employment of nerve tonics (strychnine, veratrine) or of electricity, is not promising.

4. Obstruction of the Esophagus. Obstructio oesophagi.

(*Foreign Bodies in the Esophagus; Occlusio Oesophagi.*)

By obstruction of the esophagus is meant a sudden closure of its lumen by morsels of food or foreign bodies.

Etiology. Obstruction of the esophagus is seen most commonly in cattle and is caused by large, solid constituents of the food (potatoes, beets, apples) or exceptionally by entirely foreign bodies (see foreign bodies in the stomach). Sometimes unusually large morsels of rough fodder may remain wedged in the gullet. Sometimes also an esophageal sound which has been introduced with insufficient dexterity may be swallowed and may remain wedged in the gullet. Only exceptionally hair balls or other foreign bodies of this kind may be displaced from the rumen into the esophagus during rumination. In sheep the esophagus may be obstructed by firm parts of food (beets) or by large morsels.

In horses obstruction occurs by coarse and dry feed or by large morsels, if they feed greedily and do not masticate the fodder properly and mix it with saliva, or when the reflex irritability of the esophageal nerves has been diminished by a previous narcosis. More rarely the esophagus of horses becomes obstructed by foreign bodies, such as eggs, pills, broken-off pill sticks, sounds, swallowed milk teeth, etc. Multiple occurrence of obstruction of the esophagus has repeatedly been observed after feeding dried turnip chips either alone or together with oats, since such turnip chips require much mucus to facilitate their being swallowed and hence may easily become lodged in the esophagus.

In swine, the affection occurs quite often and the obstruction is caused by pieces of potatoes, fruit, leaves, etc.

The esophagus of dogs may become obstructed by parts of the food (bones, cartilages, large pieces of meat or sinews, fishbones) or by foreign bodies mixed with the food, or by objects swallowed in play.

Exceptionally a foreign body that has been swallowed may get into the esophagus in vomiting and may become wedged in its wall (Zietschmann).

In horses, foreign bodies are usually found in the thoracic portion of the esophagus, in other animals most frequently directly behind the pharynx or in front of the thoracic aperture.

Pathogenesis. When parts of the food or foreign bodies become wedged in the esophagus, they usually cause a complete obstruction of its lumen, in very exceptional cases a thin wedged-in foreign body may cause a more or less incomplete stenosis of the lumen. Total obstruction prevents deglutition and the expulsion of the gastric contents (as in rumination, eructation, vomiting). After partial obstruction by thin bodies, fluid, gaseous, or thin mushy matters may still be transported in either direction. Whenever a foreign body becomes lodged in the esophagus, it causes convulsive contractions of the esophageal muscularis and pain while the spasm lasts. The animal experiments of Kahn and Meltzer have shown that the convulsive muscular contractions are the more frequent and the more energetic and of longer duration, the nearer to the cardia the obstruction is situated. In this manner lodgment of a foreign body in the most caudal portion of the esophagus causes a constant muscular spasm, while obstruction in the first portion of the esophagus leads to less intense muscular contractions, or they may be entirely absent, while sharp and pointed foreign bodies produce continuous pains in consequence of trauma to the wall. Stretching of the wall of the esophagus by the foreign body itself or by the swallowed material subsequently causes antiperistaltic movements.

Pressure of the foreign body upon the neighboring organs disturbs their function, more or less, while the compressed or directly injured mucosa of the esophagus may get into an inflammatory condition which may lead to necrosis. Inflammatory changes frequently are also produced in front of the obstruction in consequence of accumulating, decomposing materials. This diminishes the resistance of the esophageal wall and makes an acute dilation of the esophagus possible.

Symptoms. Pain and the peculiar sensation produced by the lodgment of the foreign body cause the animal to make strong efforts at deglutition, and to become quite restless. The animal suddenly ceases to feed, becomes restless, lowers and stretches its head and makes strong attempts at deglutition, now and then it opens its mouth, from which saliva flows abundantly; the expression of the face betrays great anxiety, the tongue protrudes from the mouth, and a convulsive cough is heard from time to time. The symptoms eventually become less marked, but are liable to recur now and then. The ingestion of food and drink is entirely abolished or the patients attempt to swallow food and water after becoming more quieted and on feeling hungry. But these are soon thrown out of the mouth and nose, although in incomplete obstruction water may get into the stomach. These attempts give us some information about the site of the obstruction. If it is located in the anterior portion, morsels of food and water at once return after an attempt at deglutition; if the obstruction is at the caudal end

we may see how the swallowed material passes along the esophagus as indicated in the left jugular depression. The expulsion occurs the later the nearer to the caudal end the obstruction is located. The patients sometimes keep on swallowing in spite of the obstruction, until the esophagus is filled completely, even up into the pharynx; then the ingestion of food usually ceases, but the animals continue to masticate on an empty mouth and to make convulsive attempts at deglutition. In such cases the gullet can be felt as a firm or more or less soft cylindrical mass on the left side of the neck. Kneading of the mass may eventually excite gagging. From time to time masses of masticated food mixed with saliva are expelled from the mouth and nose; during these attacks the animals are quite restless, and the mass expelled is undigested and does not contain any free HCl. If the cervical portion is obstructed, a circumscribed swelling appears, as large as a fist, in the left jugular groove, but may extend to the right side. The swelling is firm or hard, or it may be soft; in time it becomes sensitive to pressure, which produces gagging or the rejection of chewed food. After obstruction has lasted some time, dilation of the esophagus sometimes occurs (see page 66). In hogs there is no circumscribed swelling on the neck, at most a diffused enlargement with higher temperature, which is produced by perforating foreign bodies.

In cattle and in ruminants in general, meteorism is produced the more rapidly, the more complete the obstruction and the more fermentable the previously ingested food has been. Gases can, however, be expelled partially in incomplete obstruction of the esophagus, hence meteorism is only moderate. Under these conditions the animal may begin to feed and then produce a complete obstruction by the swallowed morsels which fail to pass down.

If the obstruction lasts for any length of time in hogs, they may become moderately bloated; in such cases they stand quietly, with their head bent down, their mouth open; they are salivated, gagging occurs as if they wanted to vomit. They do not lie down, or if they do at all, for a short time only. Thirst is increased, the animals try repeatedly to drink, but the water returns to, and runs out of the mouth. If the foreign body is near the pharynx, the animals produce a yelping sound instead of a grunt.

In a dog, Gaucet saw edematous swelling of the head, probably on account of compression of the jugulars by the foreign body (rubber ball).

Course and Prognosis. Foreign bodies lodged in the esophagus are frequently and often expelled within a short time in consequence of repeated gagging, or they are transported into the stomach or rumen by continuous strong contractions of the muscles of the esophagus. In this manner the animals recover

[illegible]sly. This happens the more easily, the smaller, smoother and softer the foreign body is and the nearer it has become wedged to either the anterior or posterior extremity of the esophagus. However, if the efforts of the patients are futile and if they are left to themselves, there occurs a progressive deterioration of their condition. Cattle may die from suffocation within a few hours after the obstruction occurred, in consequence of the rapidly developing bloating, followed by disturbances of respiration and circulation. A rapidly fatal issue may also take place in some cases in consequence of compression of the trachea or of the nerves running along the esophagus, or of obstruction of the larynx by particles of food (Sequens).

In other cases, the animals may suffer from an obstruction for some time, even for several days, but in such cases progressive and rapid emaciation becomes noticeable and the wall of the esophagus which is compressed by the foreign body becomes necrotic. If the necrotic portion of the wall breaks into the surrounding tissues, the clinical picture terminates by a purulent or ichorous inflammation of the cervical connective tissue or, if the thoracic portion of the esophagus is involved, by a pleuritis.

Obstruction caused by morsels of food may, without veterinary interference, be recovered from speedily, i. e., if the morsel becomes softened and if it can then be removed. If, on the other hand, the morsel dries and becomes harder, it may produce dilatation or perforation with grave consequences. A fatal issue may, however, occur occasionally after removal of the foreign body into the stomach, if artificial feeding has been neglected (Drouin) or if an intense inflammation of the esophagus has occurred and now of itself causes intense difficulty in deglutition (Johne). Difficulties in deglutition and vomiting may, especially in the horse, lead to pulmonary gangrene from the start. In greedy horses, obstruction of the esophagus by too large morsels may recur repeatedly within a short time.

(Small, pointed bodies frequently perforate the wall of the esophagus and become encapsulated in the neighboring tissues without producing any marked disturbances [Bruckmüller, Kitt].)

Diagnosis. In obstruction of the esophagus by lodgment of a foreign body we can usually get a proper clinical history and we have the sudden appearance of the grave disturbances of deglutition. In some cases, particularly if the obstruction is in the thoracic portion, the diagnosis may meet with considerable difficulties. The cause and site of the obstruction may be ascertained in a number of cases. Pieces lodged in the anterior portion of the esophagus may be seen in the well illuminated pharynx of the dog and cat or they may be palpated with the

finger; in cattle they may be felt after the mouth has been opened, and the whole hand introduced.

If a foreign body has lodged in the cervical portion of the esophagus, the jugular depression is only bulging at a circumscribed space, either at the left or at both sides; in the latter case the bulging is more marked on the left than on the other side. By palpation one may occasionally be able to determine the consistency and shape of the body; this manipulation usually causes gagging or the expulsion of a foamy fluid.

The location of an obstruction in the thoracic portion can only be ascertained by the use of the esophageal sound or by a careful analysis of the disturbances of deglutition. Foreign bodies of a high specific gravity (especially metals), may be made visible in all animals by the aid of the Roentgen (X) rays.

Spasm of the esophagus can be distinguished from the milder rapidly recovering cases of obstruction by the use of the sound, which must sometimes be preceded by the administration of narcotics (see page 51).

Stenosis or dilation of the esophagus may be excluded from the clinical history and from the observation, that soft feed and, still more, fluids will pass the gullet without difficulty some time after feeding. In paralysis of the esophagus, forced attempts at deglutition and gagging are absent.

In ruminants, acute meteorism, due to some other cause, might erroneously be referred to obstruction of the esophagus; but if not due to the latter condition, there is no disturbance and no regurgitation, and the sound meets no impediment in the esophagus, except in ruminants; masses vomited from the stomach, smell sour, contain free HCl, and true vomiting occurs within a certain interval after feeding and with participation of the abdominal muscles. In dogs the possibility of rabies must be considered, since disturbances of deglutition are so common in this disease.

Treatment. Removal of a foreign body wedged in the first portion of the esophagus is best accomplished by the hand or by a suitable instrument introduced into the buccal cavity.

For this purpose the head of cattle is stretched forward, the mouth is opened by the aid of the mouth gag or simply by drawing the tongue out and to the side, next the right hand is introduced into the pharynx and the foreign body is withdrawn by the fingers of the operator. Since attempts at deglutition are frequently caused by this manipulation, and since the former might move the foreign body towards the stomach, it is best to have an assistant fix the latter from the outside. The removal of the foreign body may also be brought about in such a manner that the fingers introduced into the esophagus are spread out, the assistant pushes the obstruction towards the buccal cavity and lowers the head of the animal at the same time. In this manner the foreign body usually slides between the fingers into the palm of the hand of the operator (Rolfes). The procedure is usually applied to the standing animal; it becomes necessary only exceptionally to have the animal lie down; in such a case it should lie on the right side; horses must be thrown. Foreign bodies in the first portion of the esophagus of smaller animals are best removed by appropriate forceps.

Since the animals get excited during this manipulation and since the hand introduced into the pharynx interferes with respiration, it is necessary to act

[illegible] If the procedure has not been successful, it is advisable not to prolong the attempt but to wait for some time and then try again. Such repeated attempts are however not without danger and Denenbourg has seen an inflammatory cervical edema follow them, which disappeared only after one month.

When the foreign body is located in the cervical portion of the esophagus, one should always attempt to dislocate it towards the buccal cavity unless there is danger of suffocation.

For this purpose the operator grasps the neck of the animal at the sides from above with both arms, and approaches both hands immediately behind the foreign body. He tries to move it forward by alternate pressure, made best with the thumbs. If the procedure appears to be successful, it is continued until the foreign body has been pushed in front of the larynx. It must then be fixed in this position by an assistant, to be removed through the pharynx as described above. This is sometimes not necessary, because the animal begins to gag and expels the foreign body through the mouth without any further aid. To assist in the removal of the foreign body, it is advisable to press it and the larynx forward and downward and to lower the head and approach it to the thorax before the hand is introduced into the mouth. This causes a depression of the root of the tongue, and an enlargement of the space between it and the soft palate so that the foreign body can fall out more easily (Martin). It is also well to open the mouth as much as possible (Favreau). Chapellier uses two devices similar to hoof-forceps; with one he compresses the jugular gutters behind the foreign body to prevent its sliding backward; with the other he presses upon the posterior end of the foreign body and pushes it upward. The two forceps are alternately moved forward until the foreign body has been brought into the pharynx. Imminger removes foreign bodies in the most anterior portions of the esophagus by introducing a small trochar into their center from the left side of the neck: with this he then moves them up gradually. Mord uses esophageal forceps with ring-shaped end piece, the inner surface of which is provided with hooks. This is pushed into the gullet and grasps the foreign body which is steadied by an assistant.

Reiche places a loop of rope around the neck of the animal and then passes the free end of the rope over a barn joist or limb of a tree. The animal is placed directly under this point and the loop adjusted so that it passes around the neck of the animal just below the foreign body. Two men now pull at the free end of the rope as if to lift the animal straight up. The noose thus being drawn tight glides toward the head, thereby pushing the foreign body into the pharynx, from where it can easily be removed by the hands.

Some cases can be treated successfully by the subcutaneous injection of certain medicines. Hogs and dogs may receive an emetic subcutaneously (hogs, 0.02-0.03 gm. veratrine or 0.05 gm. hydrochlorate of apomorphine [Moulis], dogs, 0.01-0.02 gm. apomorphine). These emetics sometimes cause the expulsion of the foreign body after a few minutes. In other animals likewise, the following subcutaneous injections may accomplish the object, even after unsuccessful attempts at extraction, by causing energetic muscular contractions and powerful attempts at deglutition which move the foreign body into the stomach, viz: Strychnine (Maury), eserine (Clerc), veratrine (Michalski), arecoline (Fröhner).

In cases where he did not succeed in removing the foreign body, Schaak proceeded as follows: He poured one quart of mucilaginous fluid into the animal and then made it run uphill; in this manner horses and cattle often swallowed the foreign body.

If the methods described are unsuccessful, the further procedure depends upon the nature of the wedged-in body. Fruit,

bulbs, morsels of food, etc., in the course of time become softened in the esophagus and are finally swallowed. Since obstruction of the esophagus in cattle soon leads to bloating, one may leave the foreign body undisturbed only if meteorism has been relieved in the meantime. It is therefore necessary to perforate the rumen and it is best to leave the shield of the trochar in the wound, closing its outer opening with a cork, and to open it from time to time in order to let out the accumulating gases. It is also necessary, in the further course of the disease, to attend to the artificial feeding of the patient per rectum.

If the wedged-in body is too hard, or if its nature is unknown, if one wants to accomplish removal when other means have failed, displacement towards the stomach may be attempted by the aid of an esophageal sound, a catheter, and in an emergency, with a carefully wrapped elastic stick (stick of a whip, piece of rattan), or a piece of moistened and oiled rope with a knot at one end. The foreign body must, however, be pushed without much force and very gradually. The extraction of foreign bodies with special armed extraction forceps or the crushing of the foreign body is not to be recommended.

If the removal of the foreign body in the cervical portion of the esophagus is impossible by any method above described, or if these methods are unpromising from the start on account of the nature of obstruction, the division of the foreign body with the aid of a tenotome may be attempted in the following manner: The foreign body is pushed to the left side, an incision is made into it with a fine-pointed tenotome, next a dull-pointed tenotome is introduced and the division of the body is attempted (Imminger). In some cases, however, esophagotomy cannot be avoided. Porcher and Morey removed a foreign body (teaspoon) from the caudal extremity of the esophagus of a dog by a laparo-gastrotomy.

Obstruction of the esophagus by dried turnip chips can be prevented by soaking them in 2 to 3 volumes of water, or by the liberal addition of chopped roughage, thus necessitating a better mastication and a greater admixture of saliva.

5. Narrowing of the Esophagus. Stenosis oesophagi.

(*Verengerung des Schlundes* [German].)

Etiology. Stenosis of a shorter or longer section of the esophagus may be brought about by cicatricial contraction of the mucosa (strictura oesophagi), caused by deeply penetrating inflammations or injuries, crushing by pointed or rough foreign bodies, destruction of tissues by corroding poisons. Thickening of the mucosa or of the muscularis of the esophagus produces a similar effect, likewise an abscess or a neoplasm or other

swellings, such as carcinoma, actinomycosis, papilloma, nodules due to spiroptera sanguinolenta. In the case of a cow seen by Joest, a pediculated, nodular, spindle-celled sarcoma of the lower end of the esophagus always receded like a ball valve during deglutition, to be again pressed into the esophagus upon the filling of the rumen. In the case of a horse reported by Schimmel a single gastrus larva, which had penetrated into the esophageal wall, produced stenosis; while in a case reported by Vion a mass of about 350 **Gastrophilus** larvæ in the cardia portion of the stomach and partly projecting into the cardiac opening of the esophagus caused stenosis of the esophagus followed by dilatation.

Stenosis of the esophagus in the horse may be due to continuous and often repeated spasm of the cardia. The occurrence of muscular hypertrophy with dilation of the esophagus, but in the absence of any obstruction (Kitt, Fröhner and others) to which one could refer the hypertrophy and the subsequent dilatation of the esophagus, permit the conclusion that stenosis may be due to the causes given above. This is also shown by an observation of Woodruff who failed to find anything abnormal in the esophagus at the post-mortem examination of a horse which, during life, had presented the symptoms of esophageal stenosis and which, after the introduction of the finger into the cardia, had always shown strong antiperistaltic movements. Petit and Germain are, however, inclined to look upon muscular hypertrophy at the caudal end of the esophagus as depending upon chronic dilatation of the stomach, due to abnormal gas formation.

Compression of the esophagus is frequently due to neoplasms (melanoma, struma, lymphoma, myoma) exostoses, tuberculous masses in cattle, but also in dogs, tuberculous mediastinal glands, purulent or tuberculous peribronchial glands; sometimes also to arteries with an abnormal course (Labat), to echinococci in the liver (Weber), to a greatly enlarged spleen in cattle (Vroemen), or to tuberculous lesions in the liver. All these formations may cause lateral or annular compression and diminution of its lumen (Compressio esophagi).

Cases of stenosis in a circumscribed place due to anomalies of development are very rare (Casparini and Serres saw such a case each in a foal, Smith in a one-month-old dog).

Pathogenesis. With the exception of the congenital stenosis or of cases due to spasm of the cardia, all other forms develop very slowly and the symptoms therefore rarely appear suddenly or immediately after birth, or after the weaning of the young when these are fed with vegetable food; but they make their appearance very gradually. Since inhibition of the passage of morsels of food stands in direct relation to the degree of obstruction, it may happen that a larger or firmer mass of food becomes caught accidentally in front of the obstruction. As long as arrested it will cause the symptoms of obstruction

of the esophagus by a foreign body (see page 54). The convulsive contractions of the muscles of the esophagus, caused by the arrested morsel, press the latter through the narrowed place after a shorter or longer time, and deglutition is then again possible. Repeated and strong efforts at deglutition made voluntarily by the animal also assist in pressing the morsel through the stenosed portion.

Symptoms. The symptoms consist from the start in disturbances of deglutition. The animals begin to feed greedily, they masticate and swallow, and the mouthful first passes down perfectly normally, then, however, there are strong efforts at deglutition; the morsel may finally pass into the stomach. The efforts are repeated later at each act of swallowing or at each meal again and again, according to the degree of stenosis and to the nature of the feed, until the animal, half fed but tired out, desists from feeding. During the forced attempts at deglutition, saliva, or saliva mixed with masticated food is from time to time transported outwards through the mouth or nose in consequence of antiperistalsis. Deglutition of fluids, however, is at this time not at all or very little disturbed, but if the stenosis is quite marked, fluids will also cause disturbance. In some cases, there is only a retarded ingestion of food accompanied for some time by more or less regurgitation.

If the stenosis increases the clinical picture of complete esophageal occlusion, or more often that of dilation, gradually develops.

A considerable stenosis in ruminants leads to chronic bloating, since belching is prevented. Bloating is frequently the first symptom in cattle (Johne) and is in certain cases, as claimed by Hamoir, not due to an obstructing impediment in the esophagus, but to pressure upon the pneumogastric nerve by tuberculous mediastinal glands. It is explained sufficiently by the impediment in the gullet.

Some information as to the seat of a stenosis may be gained from a careful observation of the animal during feeding; energetic efforts at deglutition or a discharge of saliva mixed with particles of food from the mouth or nose appears the longer after swallowing of a morsel the nearer to the stomach a stenosis is situated. The advance of the morsel may be controlled by placing the hand upon the esophagus. The best results may be obtained by the use of the stomach tube, but the use of this instrument is not always without danger. A tube of proper thickness, on being introduced into the esophagus, meets the impediment and its seat can be ascertained from the length of the part introduced. If now successively smaller and smaller tubes are introduced, the thinnest one, which finally passes into the stomach, gives the degree of the stenosis.

The introduction of this esophageal sound requires great care and some practice; it can, however, be easily accomplished on all animals, particularly in

the horse in a standing position. The anterior end of the sound is moistened with oil, glycerine, fat or vaseline, the head of the patient is elevated and so stretched that it forms one line with the neck; the mouth is opened with a mouth gag, or if nothing better is at hand with a piece of wood or cork placed between the molars, the tongue is then depressed with the left hand, which is introduced into the mouth, then the sound is grasped with the right hand, like a penholder, with the anterior end somewhat raised, introduced into the mouth and pushed carefully over the left hand into the esophagus until it meets an impediment. The sound is then in place, held for a little while, is withdrawn somewhat, and again pushed forward under even, very moderate pressure. Care must be taken not to produce a perforation of the esophagus. If the sound again meets the impediment at the same place, a stenosis at this site may be diagnosticated.

In ruminants the sound is to be introduced through the central opening of a wooden crossbar used as a mouth gag (Fig. 5). (For the introduction of the tube in horses see the chapter on acute dilatation of the stomach.) This procedure, however, must never be instituted when there are signs pointing to acute inflammation of the esophagus.

Fig. 5. Introduction of the stomach tube in cattle.

Diagnosis. Although the disease can usually be recognized from the clinical symptoms, a definite diagnosis can only be made after the use of the sound. It is also necessary to ascertain the cause of the stenosis, and in this respect the clinical history and contributory circumstances may give information. Compression of the esophagus, due to tuberculous mediastinal glands, may, ac-

cording to Johne, be recognized without fail when, aside from more or less definite symptoms of tuberculosis, there is chronic bloating without disturbance of appetite, rumination and defecation.

Prognosis. This is usually unfavorable, particularly in *cicatricial strictures or in stenosis caused by neoplasms, since* their treatment is entirely impossible or coupled with unsurmountable difficulties. In long standing stenosis, the nutrition of the animal is more and more disturbed, and if the animals are not slaughtered they perish from inanition, or the wall of *the esophagus ruptures at the dilation, anteriorly to the stenosis.* This again leads to ichorous inflammation of the connective tissue or to pleuritis. (Such a course has been observed by Sequens in a case of esophageal stenosis due to a spiroptera tumor). Aspiration pneumonia is a frequent and dangerous complication and may arise at every stage of the disease.

Treatment. If stenosis in the cervical portion is due to a pediculated or an encapsulated neoplasm of the esophagus, operative interference may eventually bring about recovery. A cicatricial stricture may be amenable to gradual dilatation by increasingly larger sounds (elastic rods, handles of whips, moistened ropes, catheters). This procedure can, however, rarely be carried out in veterinary practice since it requires a long continued course of treatment. If the animals are for some reason or other to be kept alive, one must attempt to retard the course of the disease by feeding soft and fluid nourishment.

Stenosis due to actinomycosis may be treated with iodide *of potash (6-10 gm. daily) iodipin, iodine-vasogene.*

In a case of Strebel the tube introduced into the esophagus of a cow met an impediment which was finally overcome, whereupon pus was discharged abundantly; the animal recovered. (Abscess of the wall of the esophagus.) A similar case has been described by Guilmot and Rheinheimer.

6. Dilatation of the Esophagus. Dilatatio Oesophagi.

(*Erweiterung des Schlundes* [German].)

Dilatation of the esophagus is a *permanent morbid condition* in which the lumen of the esophagus is either uniformly enlarged (ectasia oesophagi) or in which the wall is dilated at a circumscribed spot (diverticulum oesophagi).

Occurrence. Dilatation of the esophagus is a rather rare occurrence; it is observed principally in horses in the lower cervical or in the thoracic portion. It is considerably more rare in cattle and occasionally presents in this animal numerous wart-like excrescences. In other domestic animals the disease

occurs only very exceptionally (Schellenberg and Cadéac have each reported one case in a dog).

Etiology. Dilatation of the esophagus usually develops gradually, being secondary to long continued esophageal stenosis, after the muscularis of the part anterior to the stenosis has become weakened, so that food collects here. Sometimes occlusion of the esophagus may lead to dilatation which may make its appearance within two to five days (Maury, Pr. Mt., authors' observation), i. e., when the muscular coat of the esophagus has been weakened by inflammation. The ectasias are sometimes spindle-shaped, sometimes cylindrical, more rarely sacculated and their wall, at least in its greatest portion, is formed by all layers of the esophageal wall.

More rarely and as a rule in older horses dilatation develops as a primary affection. This might be due to a diminished elasticity of the wall of the esophagus in advanced age; in other cases psorospermosis of the muscle (Friedberger & Fröhner), or disease of the pneumogastric nerve (Kitt) may play a rôle. The hypertrophy of the muscularis, however, which is present in cases of this type, points to a previously existing impediment to the passage of food, which may have been caused by disease of the pneumogastric nerve with spasm of the cardia (see page 60). Ectasias of this type extend over a large portion of the esophagus.

Schimmel saw an ectasia in a horse with profound paralysis of the recurrent nerve which was 58 cm. long. He considered the affection to be due to gastrophilus larvae which had located there while the animal was still a foal.

Basing his views on a carefully investigated case, Kelling claims that dilatation without stenosis is due to atrophy of the longitudinal muscle fibers of the esophagus, this in its turn being caused by degeneration of fibers of the pneumogastric nerve following infection.

Diverticula of the esophagus are produced by pressure from the lumen, especially by foreign bodies or food material lodged in the esophagus or by tumors growing in the esophagus (diverticulum per pulsionem ortum), in which case they produce in animals a so-called false diverticulum, especially a hernia of the mucous membrane (esophagocele). In the latter condition the mucous membrane is pushed out through a cleft in the muscularis, which is formed by a separation of bundles of muscle fibers due to overexpansion after partial rupture, exceptionally through congenital clefts (Joest). More rarely a portion of the esophagus is drawn outward by heavy pendulent neoplasms attached to its external surface, by enlarged and later contracting diseased lymph glands attached on the one hand to the wall of the esophagus, on the other hand to the trachea or a bronchus, or by contracting connective tissue taking the place of abscesses in the vicinity of the esophagus (diverticulum per tractionem ortum).

Diverticula are sometimes short, sometimes they run as long parallel tubes above or laterally to the esophagus with

which they may communicate by a wide opening or by a narrow cleft.

Pathogenesis. Food collects in the ectatic esophagus or in the diverticulum at each feeding; water likewise collects if the diverticulum is directed downwards. This finally becomes filled completely and then compresses the esophagus so that it produces stenosis or even complete obstruction. The morsels which now follow, either pass with difficulty or they become wedged in. An ectatic place, situated in the first portion of the esophagus, is less serious if there is no stenosis back of it, since strong contractions of the muscles of the pharynx suffice to press food and drink down into the cervical portion of the esophagus.

The impaction in the ectatic portion of the esophagus creates a feeling of fullness and causes repeated convulsive contractions of the muscles immediately in front of the dilated portion and also of those within it if it contains muscle fibers.

The convulsive contractions produce a feeling of intense pain, but after more or less time they finally expel the contents of the dilated portion either into the stomach or towards the pharynx. If the latter is the case, the transport has been aided by anti peristaltic movements. Dilatation of the esophagus in consequence of pressure more or less interferes with the proper function of neighboring organs.

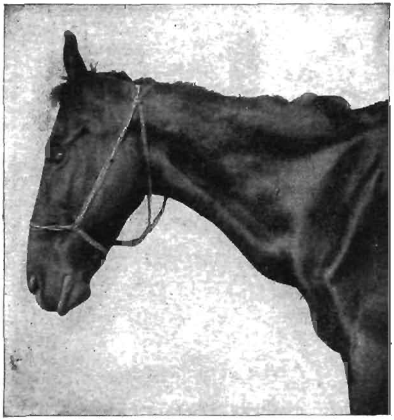


Fig. 6. Ectasia of the esophagus in a horse.

Symptoms. The animal begins to take up food greedily; however, it desists after a little while evidently satisfied, withdraws from the crib and as in obstruction or stenosis makes empty masticatory movements with lowering of the head and convulsive contractions of the muscles of the neck; smacking noises

are also sometimes heard. All these symptoms are sometimes mild, sometimes intense. They are usually more marked when rough food is taken than during the ingestion of cereals or water. (In a case of Arloing a donkey showed distress only when drinking water.) With the above symptoms one sees that the patients occasionally vomit undigested food mixed with saliva, and there may be a nasal discharge of mucus mixed with particles of food. In this, as in other forms of obstruction of the esophagus, horses sometimes exhibit a mild colic. Zürn has seen periodical contractions of the esophagus running from the thoracic to the cervical portions.

By repeated attempts at deglutition the animal finally succeeds in transporting the contents of the dilated portion, *either towards the stomach or to the exterior; then the ingestion of food is again resumed.* (During one period of feeding several interruptions of this kind may be noted.) The patient may in this manner take up an entire ration, although much more slowly than normally. In those milder cases where the ectatic portion temporarily becomes partially or *completely empty, disturbances in the intake of water are* usually seen only directly or shortly after the ingestion of solid food:

Diverticula may exist without any marked disturbances of deglutition (Zürn), they may lead to only retarded ingestion of food with a discharge mixed with particles of food. The clinical picture here is similar to that of stenosis of the esophagus. The condition may become obvious only upon the occurrence of complications (aspiration pneumonia). In a small pig. F. Mueller observed insufficient development of the body, frequent coughing, shortness of breath and throwing up of very small quantities of food material.

If a dilatation exists in the cervical portion of the esophagus, *one sees a more or less circumscribed, roundish, or* more cylindrical swelling in the left jugular depression, which may or may not extend toward the right side. The former condition is rather rare. The swelling is usually doughy, more rarely firm in consistency; it gives a tympanic souud before the ingestion of food, becomes larger after it, and it can be *made to decrease in size by pressing or kneading; simultaneously* with such manipulations there is a discharge from the nose of a slimy, eventually fetid mass of masticated food. These manipulations sometimes also cause gagging. The nutrition always suffers, generally from the start; progressive emaciation indicates some internal trouble at a time when the symptoms are not yet well marked. The more the difficulties in deglutition increase, the more the animal emaciates, and it may in fact finally starve to death.

In ruminants there is also present a chronic bloating due to the obliteration of the esophagus, which is at first temporary then continuous.

In the later course of the disease, pressure upon the trachea, *the bronchi and the pneumogastric nerve after food ingestion* produces disturbances of circulation and respiration; patients may occasionally suffocate in consequence of pressure (Friedberger & Fröhner) or in consequence of the aspiration of food material.

Course. With an increase of the stenosis behind the dilatation, or with an increasing loss of power of the muscular coat the propulsion of the contents becomes more and more incomplete in spite of stronger efforts at deglutition. The patients cease to take food much sooner, because they become tired out earlier, and they leave more and more food untouched. When the occlusion has become complete the ingestion of food and water becomes impossible, or the wall of the ectatic portion becomes gangrenous, or it ruptures with the production of fatal ichorous inflammation.

Rupture of the dilatation or the diverticulum manifests itself in a sudden aggravation of the condition, the pulse becomes rapid, respiration forced, the temperature rises, and symptoms of ichorous inflammation of the cervical connective tissue or the pleura follow; ingested water and food can now get directly from the esophagus into the pleural cavity and with the symptoms of pleuritis there develops a horizontally rising area of dulness in both thoracic cavities or in one of them (Hora). The disturbances of deglutition may at any time produce an aspiration pneumonia with subsequent gangrene.

Dilatation of the esophagus, subsequent to obstruction, takes the same course as the latter. Such an ectasia has this in common with obstruction, that the former, like the latter, leads to swelling in the jugular depression if it is situated in the cervical portion of the esophagus.

Prognosis. Diffuse ectasias of the esophagus, as well as diverticula, are, as a rule, progressive affections which terminate fatally. While the prognosis is influenced by the degree of nutritive disturbance, there is always danger of perforation or of gangrene, which, of course, makes the prognosis still more unfavorable. Even if perforation should exceptionally lead only to the production of an abscess which breaks towards the outside world, the morbid changes in the wall of the esophagus and the subsequent cicatricial contractions would later on make conditions gradually worse.

Diagnosis. The symptoms presented during the ingestion of food and water suggest the character of the disease, but it can be diagnosticated definitely only when we find a temporary swelling in the neck in the jugular depression which diminishes on pressnre. This distinguishes the disease from

inflammation of the esophagus, abscesses in the cervical connective tissue (but these may develop from an ectasia), and simple stenosis from spasm of the esophagus. Paralysis of the esophagus can be excluded by its sudden appearance and short duration. A sign of diagnostic importance is the fact that the stomach tube can sometimes be easily pushed into the stomach, at other times, that is, if it slips into the ectatic portion or into the diverticulum, it is arrested and does not enter the stomach.

Treatment. Treatment can be considered only if the dilatation is situated in the cervical portion of the esophagus; in this case a narrowing may be brought about by excision of portions of the dilated wall. Collin and Schindelka have performed this surgical operation successfully. In other cases we may provide for a proper nutrition of the animals with fluid and mushy food, eliminating dry and rough feed.

(Véret in one case performed manual pressure on the cervical dilatation each time after feeding and brought about recovery within one week. Others have recommended instruments with pads which can be fastened to the neck. These could, however, be successful only in recently formed dilatations.)

7. Neoplasms in the Esophagus. Tumores oesophagi.

Papillomata, sometimes singly in the shape of small, soft, villiferous nodules on the inner surface of the esophagus, sometimes (particularly in the middle section of the gullet) in the shape of large (the size of a fist) branched pediculated soft cauliflower masses, are comparatively frequently seen in **cattle.** Sometimes multiple actinomycomata are seen on the mucosa as prominent, flat, tough or internally softened yellowish-red nodules (Siedamgrotzky, Johne) or single tumors are developed which may be as large as a fist (Jong).

In **horses** there are encountered in the esophagus fibroma (Dieckerhoff), melanoma (Roell, Besnard), sarcoma (Cadéac) and carcinoma (Lorenz), in **dogs** occasionally sarcoma, chondroma and retention cysts.

Symptoms. Tumors narrow the lumen of the esophagus in proportion to their size and consistency, and produce the symptoms of esophageal stenosis (see page 61).

One must particularly think of a tumor if there is in the left jugular depression a permanent, firm swelling, free from inflammatory symptoms, causing the sound to meet an impediment.

Treatment can only consist in the operative removal of the tumors situated in the cervical portion or, in actinomycosis, in the internal administration of iodine preparations.

8. Spiroptera Sanguinolenta in the Esophagus.

Occurrence. The occurrence of spiroptera sanguinolenta in dogs has been reported from France, Italy, China, Brazil, India, Turkestan, Japan and Tunis; 10% of the dogs of Japan

and 70% of the dogs of Tunis are said by Roger to suffer from this worm.

Etiology. **Spiroptera sanguinolenta** is a blood-red worm 3-8 cm. long; the male is smaller than the female. The tail end of the former is rolled up. The young forms of this worm exist in cysts contained in the abdominal cavity of cockroaches (Blata orientalis).

Evolution. The eggs get into the open with the feces or with the expectoration of infected dogs and are, according to Grassi, taken up by the cockroach (Blatta s. periplaneta orientalis); the larva is encapsulated in its abdominal cavity and develops further. Seurat claims that this larva is a young form of Spirura talpae, which also belongs to the Spiroptera. The larva which comes from the egg (130 μ long of Spiroptera sanguinolenta, according to him, invades bugs, batrachians, lizards, birds (chickens), hedgehogs, bats, where it passes through two moultings and attains a considerable degree of resistance. The worm is said to be mature only after the third and fourth moultings in the body of the dog.

Natural infection occurs from the ingestion of cockroaches, whereupon, after two weeks, small spiroptera become visible in the esophagus (Grassi). According to Seurat, dogs are infected by catching chickens, hedgehogs, lizards and bats.

Pathogenesis. The larvæ taken into the stomach with cockroaches wander back into the esophagus, bore into the mucosa and lead to the formation of hazelnut-sized or larger swellings the compartments of which contain up to twenty worms embedded in pus. Except for a small opening at the apex of the swellings, the mucosa appears intact. Some of the parasites frequently remain in the stomach, others may wander into the organs with the lymph or blood current, particularly into the wall of the aorta, where they likewise cause a local inflammation. According to Roger, the worms form metabolic products which excite the nerves of the heart.

Symptoms. In the more carefully studied cases intestinal disturbances were observed (painful and difficult deglutition, gagging or vomiting, empty mastication), also dry cough, dyspneic attacks, emaciation. Occasionally nervous disturbances were observed, especially weakness of the hind legs or a behavior as seen in rabies. Severe cases as a rule gradually lead to death which may also occur suddenly in consequence of rupture of the aorta. A positive diagnosis is possible during the life of the animal only by microscopical demonstration of the worms in feces or expectoration.

Treatment. This can be only symptomatic. Better success

may be had by prophylactic measures consisting in the prevention of the ingestion of cockroaches.

Other Animal Parasites in the Esophagus. There is found in the epithelial covering of the esophagus of cattle spiroptera scutata Mueller, also named Gongylomena scutatum, Leuckart, a filiform worm, 4-10 cm. long; its cephalic end is provided with shield-like chitinous scales, which are easily visible through the upper epithelial layer and whose larvæ live encapsulated in several species of dung beetles and reach the esophagus when an animal takes up such beetles (Ransom and Hall).

Larvæ of Hypoderma lineata are found during the summer and winter months in the submucous tissue of the pharynx and esophagus of cattle, where they sometimes produce a hemorrhagic infiltration without causing other disturbances. Sarcosporidiosis of the esophagus does not seem to be detrimental to health. Some authors consider it in casual relation to certain forms of esophageal dilation.

9. Diseases of the Crop of Fowl.

(a) Catarrh of the Crop. Ingluviitis.

(*Weicher Kropf* [German]; *inflammation ingluviale* [French]; *soft crop.*)

Etiology. Primary catarrh of the crop makes its appearance after the ingestion of pointed foreign bodies, of soft, scaly or, on the contrary, hard food. Richter reported numerous cases among chickens after the feeding of too dry oats. Sometimes the disease is due to the ingestion of fermenting or decaying materials (draff, distillers' mash, parts of cadavers). Too early removal of the young squabs from pigeons often causes ingluvitis in the parents, because the glands which were active during the feeding of the young birds may subsequently become inflamed.

Secondary ingluvitis is caused by animal parasites and is seen in thrush.

Symptoms. The animals lose their appetite, are listless, stretch their necks repeatedly and swallow with difficulty. The crop is tender to pressure. The fermenting contents cause a marked protrusion of the region of the crop, the latter feels soft and is tympanitic on percussion (socalled soft crop). Fetid gases escape from the beak upon pressure on the crop or are occasionally brought up by vomiting; the fluid contents are likewise emptied and they smell disagreeably sour.

These escape through the beak and partly through the nose. Disturbed nutrition leads to emaciation; the animals become very weak and frequently die. After a long continued affection of this kind or after repeated attacks a socalled pendulous crop develops frequently.

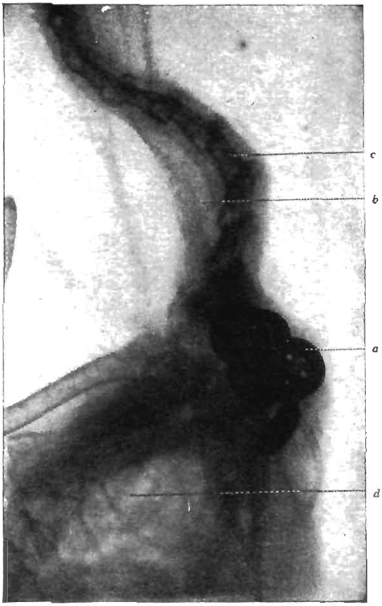


Plate I. *Foreign bodies (metal buttons) in the crop* of a hen. a. Metal buttons in the crop; b. trachea; c. cervical vertebra; d. lung (Röntgenograph).

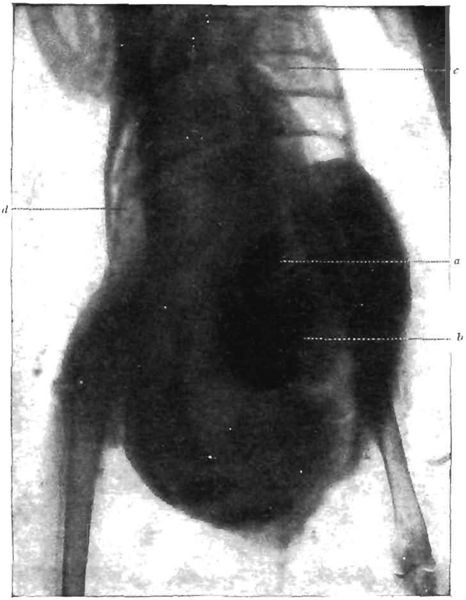


Plate II. Foreign bodies (metal buttons) in the stomach of a hen. a. Two metal buttons in the proventriculus; b. several metal buttons in the gizzard; c. lung; d. air-sac (Röntgenograph).

Treatment. One must particularly try to empty the crop of its contents by holding the animal's head down and carefully kneading the crop towards the buccal cavity. The next step consists in the administration of mild disinfectants (2% boracic acid solution, 1% sulphate of iron) or astringent solutions (alum, tannic acid, juice of lemons); peppermint tea or fennel water to which 10% hydrochloric acid has been added is also serviceable, also a 5% solution of Carlsbad salt (Klee), 0.1% solution of salicylic acid as drinking water. The animals are starved for a day and on the following days allowed a smaller quantity of food. In very obstinate cases irrigation of the crop may be instituted with good success.

Irrigation of the crop, according to Klee, is performed as follows: A funnel is placed at the end of a thin rubber tube 40 cm. long; the free end of the tube is then introduced into the crop through the beak. The funnel is now filled with a 1% solution of boracic acid, it is next elevated and the solution is allowed to run into the crop. Finally the funnel is lowered and the fluid is permitted to run out. While it escapes the crop is carefully kneaded or massaged.

The disease can sometimes be cured only by an incision into the crop. This operation also seems indicated in pendulous crop, provided the catarrhal condition has already disappeared.

Catarrh of the crop in pigeons due to the early removal of their young ones may be cured rapidly by substituting other young ones.

(b) Occlusion of the Crop. Obstructio ingluviei.

(*Hard crop, indigestion ingluviale* [French].)

Etiology. Occlusion of the crop is frequently due to overfeeding, especially with dry grains (millet, corn, peas, oats) or with a feed containing much bran or chopped straw (peas, oats, pumpkin seeds, wild grapes) after a long period of starvation, or to unusual conditions, most frequently at the beginning of a period of harvest, especially in chickens, which otherwise are also more disposed to this disease. Bearded barley and oats still attached to the straw are most injurious to geese and ducks in this respect, particularly when there is no water in the vicinity to wash it down (Eber). The consumption of large quantities of sharp roughage mixed with bran may also prove dangerous, especially if the birds have previously been kept on a scant diet (according to Eber as a result of the war). But also the feeding of too large quantities of feed mashes can produce an occlusion of the crop by provoking a relaxation of the wall of the crop, particularly in chickens, which naturally are grain-eating birds. Ducks and geese, more seldom chickens, suffer from this trouble after eating large quantities of dry grass in the spring and fall when there is a lack of other feed; the grass easily forms matted balls in the crop (Eber). Hébrant and

Antoine observed in ducks and sometimes in swans repeated cases of occlusion of the crop, frequently with a simultaneous occlusion of the glandular and even of the muscular portion of the stomach, in the winter, caused by eating woody or tough plants when other feed was scarce. In water fowl the disease follows upon the ingestion of certain aquatic plants (chara, cynodon, triticum repens, corex) or of ailanthus glandulosa. Young pheasants sometimes contract the disease after the ingestion of numerous insect larvæ. Sometimes foreign bodies (pieces of metal, stones, pieces of glass, pieces of bones, fragments of egg shells) or many small stones cause the disease. Unusually large pieces (pieces of potatoes, turnips or carrots, potato and apple peelings, etc.), unminced sausage skins, slaughter offal, intestines from slaughtered poultry and rabbits may cause the disease (Eber). In one case in a pigeon reported by Eber the occlusion of the crop was caused by a cheesy focus breaking through from the trachea to the esophagus, and exceptionally they may be produced by animal parasites (Railliet and Lucet).

Symptoms. Obstruction of the crop manifests itself by the fact that the animal becomes listless, it repeatedly opens the beak and the latter sometimes discharges an ill-smelling fluid. *The appetite is absent either from the start, or after some time* if the obstruction is due to a foreign body. The crop appears enlarged; its wall is tense, elastic, sometimes doughy, sometimes hard in consistency (socalled hard crop), depending upon the mass of accumulated food and upon the amount of gases that have formed. Foreign bodies in the crop may be felt through its wall or may be demonstrated by a Roentgenograph. (See Plate I.)

Course. Obstruction of the crop rarely disappears spontaneously. If nothing is done, chickens usually die of exhaustion after a few days; water fowl, however, succumb to suffocation after a few hours (Dupont). This difference in the course can perhaps be explained by the fact that the crop of chickens is really an extrusion directed outward, while the crop of water fowl is a circular dilation of the esophagus, which may easily compress the trachea. Occasionally rupture or perforation of the crop occurs. Rivolta and Delprato saw the formation of an enormous crop (socalled pendulous crop) in a hen (Kitt).

Treatment. If the contents are not too hard, massage of the crop, and kneading of the contents towards the beak or under certain conditions irrigation of the crop are indicated, while in impaction of the crop, or if massage should prove unsuccessful, incision of the crop must be resorted to. In order to prevent the trouble, offal of animal origin should only be fed boiled or

cut into small pieces and grain should be fed in small quantities. In winter, spring and late autumn and during scarcity of feed proper additions should be made to the feed.

Incision into the crop is comparatively simple and without danger except in pigeons. The feathers are removed with scissors in a region about two fingers wide and one finger long. Then one makes a pointed incision in the middle of the crop and prolongs the wound parallel with the long axis of the neck from 1 to 1½ cm. If the contents of the crop consist of small pieces they can now be removed *by kneading; if larger leaves are present they can be removed by the aid of* a pair of forceps. The wound is then irrigated and sutured. It usually heals rapidly and the stitches can be removed in five to seven days. On the first day after the operation the animal must not receive anything except stale white bread soaked in claret; after thirty-six hours abundant water, but only a small amount of soft food.

(c) Animal Parasites in the Crop.

Dispharagus uncinatus (Filaria s. Spiroptera uncinata) lives in the stomach and in the gullet of geese, ducks and swans (see stomach parasites). In chickens the D. nasutus and the D. laticeps have been found.

Gongylonema ingluvicola was found once in the crop of a hen by Ransom and Ciurea. (The eggs of the parasite are 50 microns long and 36 microns wide and contain an embryo with a spine at its front end.)

Trichosoma contortum is a filiform worm, 1-4 cm. long; it bores passages which show in the form of whitish or yellowish tracks in the hyperemic mucosa of the dilated gullet in ducks and geese, and seems to weaken the muscles.

The symptoms are those of severe catarrh of the crop, with rapid emaciation, narcolepsy, occasionally associated with epileptoid convulsions; after five to ten days gradual obstruction of the crop supervenes with marked increase in its circumference, and death follows one to two days later (Railliet & Lucet). During life a positive diagnosis is possible only by microscopical demonstration of the trichosoma eggs in the feces or in the wash-water of the crop after lavage.

Treatment with ¼% carbolic acid solution for drinking water, or mixed with mushy food is not very promising. On the other hand, the administration of antihelminthics may be successful (see intestinal worms). Prophylactically it is proper to keep the animals from dangerous water.

Trichosoma strumosum (Tr. delicatissimum) causes similar changes in the tissues in the gullet of pheasants as the Tr. contortum does in aquatic birds. The clinical manifestations differ only in so far as the *dilatation and obstruction of the crop are not observed.* The characteristic trichosoma eggs are found in the scrapings of the pharyngeal mucosa, in the feces and in the wash-water from the crop after lavage. The same rules are applicable to treatment and prophylaxis as in trichosomiasis of the pharynx in aquatic birds.

The **Trichosoma annulatum,** which is found in chickens, is not of clinical importance.

Section V.

DISEASES OF THE STOMACH AND INTESTINES.

1. Vomiting. Vomitus. Emesis.

Vomiting is the evacuation of the contents of the stomach through mouth or nose; it is accompanied by a sensation of nausea and occurs in consequence of direct irritation of the vomiting center (central vomiting) or, most frequently, through the reflex irritation of this center (reflex vomiting). In birds an analogous process is seen in the emptying of the crop by vigorous movements of head and neck.

The regular succession of the complicated motions in vomiting is assured by the activity of the vomiting center in the medulla oblongata. The centripetal branch of the reflex arch passes mainly along the vagus and glossopharyngeus; the centrifugal branch passes in the same nerves and partly in the motor nerves for the abdominal and respiratory muscles which take part in the act of vomiting.

Etiology. In domestic animals, vomiting always is of pathological significance and is most frequent as reflex-vomiting, particularly in the course of certain diseases of the stomach and intestines. One of the most frequent causes is overloading the stomach, especially in hogs and carnivora, not infrequently in cattle in which vomiting seems to be favored by motion. Irritation of the gastric mucosa causes vomiting after the ingestion of irritating, acrid foodstuffs (in cattle after eating green alfalfa, in sheep grazing in the Wasatch mountains of North America after eating **Helenium hoopesii**, western sneezeweed), after the administration of sharp caustic drugs and of certain emetics, also in catarrhal and inflammatory diseases of the stomach which are in part primary, or may appear in the course of acute infectious diseases (erysipelas, distemper, fowl-cholera), also in the presence of stomach worms or of gastric ulcer. Other causes are injuries of the gastric mucous membrane by foreign bodies in carnivora, in cattle and sometimes in hogs; stenosis or obstruction of the pylorus by foreign bodies or tumors; in the ruminants, occlusion of the openings between the sections of the stomach. In animals with a single stomach, vomiting, particularly after eating, may be due to compression of the stomach if tumors exist near-by. In horses, vomiting is observed occasionally after excessive drinking (Bambaner); Deli saw vomiting of this kind enzootically in horses that drank water in swamps. During continuous confinement in the barn, cases of vomiting may become frequent in cattle without apparent cause and often without digestive disturbances (Bissauge, Luginger).

Of intestinal diseases, obstruction especially causes vomiting but only in carnivora and hogs; in horses and cattle only in cases with simultaneous secondary dilatation of the stomach. In these cases the irritation is produced by the fact that the intestinal contents are rejected into the stomach and add to the accumulation in the latter; in intestinal occlusion it occurs because of twisted bowels. The sensory nerve endings in the peritoneum are irritated similarly as in the course of acute peritonitis which is frequent in cattle, due to foreign bodies in the stomach. Liver disease probably produces vomiting only by involvement of peritoneum or stomach in carnivora and hogs. It is not certain whether diseases of the internal female genital organs, without involvement of the peritoneum, can cause vomiting. Bissauge saw vomiting in a cow with prolapse of the vagina, but this was probably due to the raised position of the posterior part of the body, since it ceased immediately when the cow was put into a normal position.

An irritated condition of the pharyngeal mucosa sometimes causes vomiting in hogs and carnivora, especially in pharyngitis, after lodgment of foreign bodies, in convulsive paroxysms of coughing.

According to Dasch, vomiting often repeated up to 10 to 20 times may be observed in the bulldog while eating (especially when coarse food or food containing bones is offered), as a result of continued irritation of the mucous membrane of the pharynx, which is perpetuated in these animals by frequently breathing through the open mouth. Occasionally deep-seated inflammation of the gullet is accompanied by vomiting. Mostly the vomiting in affections of the gullet is only apparent and consists in the rejection of chewed accumulations of food, without participation of the abdominal muscles. According to Bissauge, compression of the N. vagus by tuberculous mediastinal lymph nodes may cause repeated vomiting in cattle.

In fowls the rejection of the contents of the crop may be due to catarrh and to occlusion of the crop.

Central vomiting occurs after injuries and in different diseases of the brain, especially in the region of the medulla oblongata; also in the course of uremia and cholemia, after the ingestion of certain poisons (veratrine, apomorphine, arecoline, chloroform). The vomiting observed in horses during over-sea transportation may have a like cause.

The **ability to vomit** differs in different species and also varies individually. Dogs and cats vomit most easily, then hogs, and domestic fowls as far as the emptying of the crop is concerned. The vomiting of the stomach contents cannot be produced in birds even artificially (Miller). Ruminants vomit with much more difficulty, and horses most so. Miller claims that rabbits cannot vomit at all, but according to Brown it may be observed in these animals also.

The causes for the differences in the ability to vomit are commonly attributed to differences in the anatomical relations of stomach and gullet. It is said that ease of vomiting is associated with a thin, funnel-shaped esophagus opening straight into the stomach, with the position of the latter contiguous to the abdominal wall, with a gut-like structure of the stomach, and finally with a slippery, moist consistency of its contents. As causes of difficulty in vomiting in solipeds are mentioned the oblique opening of the esophagus, the distal portion of which is supplied with thick muscles, the presence of a very strong sphincter at the cardia and of a very strong loop of the cardia-muscles; the formation of a fold of mucous membrane, which closes the cardia like a valve when the mucosa is pushed over the loose submucosa in the filled stomach, the fact that the wide, less firmly closed pylorus lies very near to the cardia, and finally the small size of the stomach.

The differences in the ability to vomit may be explained also quite readily

by differences, according to species, in the irritability of the vomiting center or of its afferent tracts. The mechanical impediments which have been enumerated can naturally only oppose the evacuation of the gastric contents to a certain degree, but not the occurrence of the visible complicated reflex movements which tend to eject the contents of the stomach. These complicated movements, which are called gagging when they occur without emptying of the stomach, would have to occur with practically equal frequency in all species under corresponding circumstances, but this is by no means the case. Eructations appear to be very frequent in animals which vomit with difficulty and in which therefore the mechanical obstacles would have to oppose the escape of stomach gas in like manner as in gagging or vomiting. The very variable susceptibility of different species to emetics and the not inconsiderable individual differences in the ability to vomit also are in support of this view. The variable irritability of the vomiting center may be thought to have developed gradually in the course of phylogeny under the influence of the conditions of life which differed according to species.

Symptoms. Vomiting is usually preceded by certain prodromal symptoms (nausea), such as slight unrest, motions with the tongue, empty acts of swallowing, eructations, salivation and changes in respiration. Hogs, dogs and cats vomit without any particular effort; the head is stretched, lowered to the floor, and the mouth then discharges part of the gastric contents. In cattle vomiting is preceded by greater restlessness and greater efforts; the animal pulls back from the manger, the fore-legs tremble, the hind legs are placed under the abdomen, the back is curved; they take a deep inspiration followed immediately by a vigorous contraction of the abdominal wall with painful lowing, after which the liquid or mushy contents of the rumen are discharged through the open mouth, rarely also through the nose. Usually large amounts of ten quarts and more are vomited at once. Horses appear anxions and excited, they tremble, perspire over the entire body, their expression is staring; presently they place the legs together under the body, draw the head in and lower the neck with a strong effort of the neck muscles; at the same time a vigorous contraction of the abdominal wall occurs accompanied by loud groaning or with a peculiar yelping sound followed immediately by the discharge of the stomach contents, mostly through the nose. Only greenish fluid mixed with saliva is usually expelled from the mouth. Vomiting is observed either as a single act or it is repeated several times or it may last for hours.

Vomiting is particularly obstinate in gastritis, in occlusion of the intestines, as well as in certain diseases of the peritoneum and of the central nervous system. The animals become the more exhausted the longer the vomiting lasts and the more frequently the act is repeated. Some animals also cough a good

In horses the vomited matter usually consists of half- or little-digested food, sometimes mixed with mucus, or it is even hemorrhagic; the mixture smells sour and has an intensely acid reaction; ruminants usnally expel the contents of the paunch only, and the expelled contents are characterized by a peculiar smell.

The vomitus of carnivora and omnivora consists likewise

of particles of food in case the stomach has been full. If vomiting has occurred several times, or if it took place on an empty stomach, the vomitus consists of a watery-mucous fluid or merely of thick tenacious mucus; the latter appears at first greenish, then brownish or greenish-yellow, because to the morbid contractions of the stomach are added the antiperistaltic movements of the duodenum. Birds sometimes expel the contents of the crop with a swinging motion of the head.

The vomitus may contain blood (vomitus cruentus) if the vomiting is caused by gastritis, gastric ulcer, a disintegrating tumor or an injury of the wall of the stomach; blood may, however, also become mixed with the contents of the stomach in the pharynx or the buccal cavity. In overloading the stomach with blood, carnivora also vomit a lot of coagulated blood. Masses of pus and animal parasites are rarely found in the blood.

Vomiting of fecal matter (ileus, miserere) may be recognized in carnivora by the peculiar color and smell if the intestinal contents have come into the stomach from the last portion of the small or from the large intestines. The absence of an acid reaction without the peculiar fecal color or smell only permits a suspicion of fecal vomiting without absolute certainty as to diagnosis.

Horses with secondary dilatation of the stomach often vomit a material similar to that of the contents of the small intestines. In intoxications the vomited matter sometimes has a smell characteristic of the poison which caused the attack (phosphorus, arsenic, hydrocyanic acid, carbolic acid).

Cases in which food masses have become wedged in the esophagus in consequence of ectasia of the esophagus and are expelled by the mouth or nose, must be distinguished from vomiting. In the former cases the vomited matter is not acid and does not contain free HCl.)

Dehne observed in an otherwise healthy cow a peculiar way of ruminating in which the cow would allow the ruminated morsels to drop out of her mouth but would eat them again when she had finished ruminating. Only when she was disturbed for a while would she leave the mass of expectorated feed.

Emetic irritation does not always lead to vomiting, but only to gagging (vomituritio). The animal shows the same behavior as in genuine vomiting, but no gastric contents are expelled.

Belching or eructation is a normal occurrence in ruminants. It is however also and more frequently observed under morbid conditions; then the expelled gases frequently have a repulsive odor. In other animals belching is always an indication of abnormal gastric fermentation.

The significance of vomiting depends, of course, upon the cause and upon the underlying disease. It is always gravest in horses where it occurs in the course of dangerous diseases of the stomach (excessive dilatation of the stomach, severe

gastritis, partial rupture of the wall of the stomach, etc.), or through an excessive irritation of the gastric nerves; in horses vomiting also gives rise to an aspiration pneumonia with comparative frequency; this is much rarer after vomiting in other animals. Exceptionally an animal may suffocate during vomiting, because the expelled feed masses may get into and obliterate the larynx or the nasal cavity (Sequens, Zschokke).

Older authors have looked upon vomiting in the horse as a sign of rupture of the stomach. It is however settled beyond doubt that these animals cannot vomit any more in total rupture of the stomach, because the gastric contents are expelled into the peritoneal cavity in consequence of the contractions of the wall of the stomach and of the abdominal press. Still, in partial rupture of the wall of the stomach an intense emetic irritation may cause vomiting. Partial ruptures are occasionally found in the stomach of horses which have vomited some days before death (from other diseases) had occurred. Rupture of the stomach may become larger or even complete after vomiting has occurred. That horses are able to vomit even if the stomach is absolutely intact has been shown convincingly by post-mortem examinations preceded by vomiting during the life of the animal.

Emetic irritation also causes a reflex relaxation of the cardia and of the esophagus, and this the authors have frequently seen upon the introduction of the stomach tube. Relaxation of the cardia may also be due to a degeneration and to a tear between muscle bundles.

Treatment. Except in horses, vomiting after overloading of the stomach and after poisoning is beneficial because the stomach becomes relieved or the poisons are removed. In such cases, therefore, it is not best to stop vomiting but this should be aided by the internal administration or subcutaneous injection of medicines (apomorphine, veratrine, ipecacuanha), or it may even be produced artificially, the emetic irritation being stopped only after too frequent attacks have become distressing. Vomiting due to other causes, and in horses all vomiting should be stopped by the application of cold (making the animals swallow bits of ice), in small animals cold compresses over the region of the stomach, and by narcotics (opium, chloral hydrate, bromine, by mouth or per rectum; morphine subcutaneously). In cattle Bissauge had good results with camphor (8 gm. with two yolks of eggs in two quarts of water). Pozajic relieved vomiting which had lasted for 4 days by the administration per os just before feeding of 2 doses of 30 gm. chloral hydrate in camomile tea with 30 gm. gum arabic. In dogs vomiting can often be stopped by administering black coffee (a wineglass to a cupful).

Holterbach arrested very obstinate vomiting in a dog by the administration of yohimbinum muriaticum Spiegel one tablet of 1 mgm. every two hours.

Aromatic medicines may also do well (such as camomile tea), also ether, ether with alcohol, camphor, chemicals developing CO_2 (bicarbonate of sodium). Valuable dogs may also receive champagne.

Vomiting of horses should always be treated by removing

parts of the gastric contents with the stomach tube (see acute dilatation of the stomach).

2. Diseases of the Forestomachs of Ruminants. (Rumen, reticulum and omasum.)

Physiology. The innervation of the different portions of the stomach is accomplished by autonomic nerve plexuses in the wall of the stomach, similar to the Auerbach plexus in the intestines, which essentially are irritated by expansion and other mechanical irritants and which on the one hand permit isolated activity of the different portions, on the other hand insure a co-ordinated functional relationship as a result of which movements in one portion of the stomach also influence the movements of the other portion. Especially will an arrested activity in one portion caused by any influence whatever also permit the other portions of the stomach to come to rest. The autonomic plexuses are subordinated to the central nervous system by mediation of the vagus as the transmitting nerve and to the sympatheticus as inhibitory nerve through the prevertebral ganglion solare and on the wall of the stomach. The movements of the rumen are only partly isolated (Marshall); for the main part they are wavelike and progress from one part to the other, whereby those of the right (ventral) sac are especially lively and strong, throwing the contents into the left sac, while through the contraction of the left sac combined with a simultaneous contraction of the reticulum, the atrium ruminis and atrium ventriculi are strongly filled and pressed against the cardia. The contractions of the reticulum take place very rapidly and simultaneously over the entire organ in the direction of the implantation of the esophagus. During these contractions the contents are almost wholly emptied and driven into the atrium ruminis. Aside from this, the contractions of the reticulum may directly continue to the abomasum and even to the omasum. Contrary to this, the contractions of the omasum are slow, sluggish, long continued and pressing, during which the rough laminæ omasi are also contracted. The movements of the abomasum are similar to those of the simple stomach, in the posterior parts more like peristalsis, and progress toward the pylorus.

Digestive Activity of the Fore-stomachs. The direct rôle played by the fore-stomachs is exclusively of motor and physical character. In these compartments there takes place a mixing, kneading, swelling, softening, extracting, dissolving and pushing away of the contents within the separate divisions. They also play an important part in rumination in that the atrium ventriculi and atrium ruminis are instruments in bringing about the return passage of the food from the rumen and reticulum. They also transport the now small, finely masticated, juicy and fluid morsels directly to the omasum by way of the esophageal groove; they carry the fluid as well as the finely divided substances (grains, bran, etc.) from the reticulum to the omasum and the fluid part of the contents of the omasum along the sulcus omasi to the abomasum. In accordance with the anatomical structure of the esophageal groove, it is opened by coarse, solid material and by large morsels as well as by greedily swallowed water, whereupon such material will fall directly into the rumen, while other material and fluids swallowed in very small quantities pass directly into the omasum and from there into the abomasum.

Indirectly the fore-stomachs also play an important rôle in digestion, as in them, with the aid of the swallowed saliva, amylases in the vegetable food material, bacteria and fermentative molds, cleavage of the carbohydrates results, with the formation of glucose and other products of fermentation (lactic, butyric and acetic acid, large amounts of carbonic acid gas and methane). Furthermore, digestion of cellulose (cracking, dissolving and splitting into carbonic acid gas, fatty acids and similar cleavage products as in digestion of starch), probably by mold-like micro-organisms (**Aspergillus cellulosæ,** according to Hopffer), and finally digestion of protein by proteolytic ferments derived from the food and by micro-organisms also takes place in them.

Classification of the Diseases of the Fore-stomachs. In accordance with the immense development and cornification of the epithelial layer and the absence of glands, diseases with changes in the tissue are only seldom observed in the fore-stomachs. Diseases of these organs consist for the most part only in disturbance of the functional activities with reference to the motor activities, the mixing, softening, splitting and fermentation of the food, especially by protein, starch, and fat-splitting plant enzymes, bacteria and molds contained in them. A classification of the diseases of the stomachs of ruminants, having full regard for the above-named conditions as well as meeting the practical requirements, will be attempted in the following:

(a) Dilatation of the Rumen. Impacted Rumen. Dilatatio acuta ruminis ab alimentis.

(*Acute Indigestion, Acute Dyspepsia* [Dieckerhoff], *Dyspepsie* [Harms], *Idiopathic paresis of the paunch* [Eber], *Pansenserstopfung,* [German]; *Indigestion du rumen avec surcharge* [French], *Empansement* [French].)

We designate as dilatation of the rumen, the filling and dilatation of the first section of the stomach of ruminants by an excessive amount of food or by dry and heavy feed which will diminish or abolish entirely the erstwhile intensified contraction of the rumen.

Occurrence. Dilatation of the rumen is one of the more common diseases of ruminants, especially of cattle. Stable-fed animals are most commonly attacked, more rarely cattle on pasture.

Etiology. The affection is usually due to the ingestion of unusually large masses of feed; it is immaterial whether the food is of the highest quality or unsuitable in character. If the animals obtain palatable hay, green feed, flour or grain feed without restriction, or if they have broken loose, they are liable to overfeed, particularly if they had been starved for some time. (There are some countries in which it is customary to increase the live weight of animals to be sold for slaughtering by feeding them excessive quantities of feed

before the transfer is made.) Animals occasionally overfeed on rich pastures, particularly if they have been fed sparingly in the barn. The affection is also brought about by feeding with voluminous feed which is too dry or improperly prepared (chaff, straw, coarse clover, hay, etc.); a low nutritive value causes the animals to ingest great quantities. Beets, leaves of beets and potatoes, particularly in a boiled mushy condition, kitchen garbage, distillers' or brewers' mash, mouldy or frozen feed may likewise cause dilatation of the rumen. Finally, lack of water (due to neglect) during dry feeding, becomes responsible for the disease.

Predisposing factors sometimes act as contributory causes, such as physical weakness, continuous stay in the stable, ingestion of too cold water, prevention of mastication by overexertion after feeding, sudden change from green to dry feeding.

Cadéac holds that dilatation of the rumen as well as acute bloating of ruminants always follows a preliminary paresis of the rumen, so that the former are always secondary conditions. Numerous observations however show that an excessive ingestion of food may bring about the affection in animals with a rumen functioning absolutely normally. One is justified in drawing the conclusion that a normally developed and normally functioning muscularis of the rumen cannot be stretched beyond a reasonable limit.

There is, however, no doubt that the rumens of different individuals differ as to the power of its muscularis, and that external conditions may exert a good deal of influence.

Eber looks upon dilatation of the rumen as a type of idiopathic paresis of the rumen in contradistinction to sympathetic paresis of the rumen due to organic affections of the different parts of the stomach and of the neighboring organs.

Pathogenesis. After the effects of one of the enumerated etiologic factors have become manifest, there exists an improper relation between the mass and weight of the ingested food and the ability of the rumen to the detriment of the latter. The consequence is that the masses of feed present in the rumen are not properly mixed and moved, or not at all, in spite of the fact that the muscularis is stimulated to powerful contractions. The contents of the rumen then swell more or less and develop, in consequence of fermentative processes, an abundance of gas. The more juicy the feed has been, the more rapidly and the more abundantly gas forms. If the feed has been too dry, gas is not formed at all, or a small amount only is formed after some time. The increasing tension of the wall of the rumen causes an increase in the power of contractions, which is painfnl to the animal. In milder cases the powerful mnscular contractions succeed in transporting a portion of the contents of the rumen, and this relieves the morbid condition. In excessive overloading, the contents do not move, however, in spite of the energetic muscular contractions, and even the escape of gases becomes impossible. After some time there occurs then a paresis of the overworked muscles of the rumen. The more, rapidly the rumen becomes dilated by masses of swollen food and gases the more respiration and circulation become inter-

fered with. The decomposition of the contents of the rumen may under certain conditions cause the formation of poisonous and irritating substances and thereby partly local inflammatory conditions aud partly general symptoms of intoxication, if such material is carried into the intestinal tract by the incompletely paralyzed movements of the fore-stomachs.

Anatomical Changes. The abdomen appears greatly dilated, the diaphragm is pressed forward, the much dilated rumen contains excessively large quantities of repulsively smelling feed *in addition to variable amounts of gas. On post-mortem* examination one finds the signs of suffocation. If the disease has lasted somewhat longer, the omasum contains desiccated masses of feed; the mucosa of the rumen, sometimes also that of the other parts of the stomach, shows diffuse reddening or hemorrhagic points.

Symptoms. The animal refuses its food; if in the open air they do not move, they place the legs under the abdomen or spread them apart, arch their back and stare. They manifest colicky pains and move the head towards the abdomen and push the latter from time to time with their horns, or they kick with their hind legs, wag their tails, groan, lie down occasionally but get up again at once. The colicky pains, while not attaining a high degree, are rarely absent. Groaning is usually heard, either only during a change of position of the body (getting up, lying down) or independently of it.

Appetite and rumination are only diminished in the milder cases but are absent from the start in the more severe type. Even in milder cases complete lack of appetite supervenes if the animals are offered a chance to ingest rough feed. The ingestion of water ceases first, although thirst seems to be increased in some cases. Occasionally the patients execute masticatory movements and foamy saliva runs out of the mouth. Repulsively smelling gases are expelled by belching, from time to time. Occasionally there occurs gagging, even vomiting, with the expulsion of much feed mash.

The abdomen is at first only moderately enlarged and the depression of the left flank seen on an empty stomach is filled out. In the further course or in excessive overfilliug, the size of the abdomen is much increased, particularly on the left side, and the left flank depression has been changed into a distension. The rumen feels doughy, firm or even hard as a stone. The same impression is created by a rectal exploration of the rumen; frequently this method makes the existing conditions much clearer than external palpation (Gebauer). The animals try to avoid firm pressure upon the rumen. At this time percussion of the left flank gives a dull and empty sound. The picture changes after the ingestion of a more or less fermentable feed, because then the left depression feels elastic on slight pressure

and gives a tympanitic sound on percussion. A firmer pressure, however, usually suffices in these cases to reveal the presence of firm masses of feed under the layer of gas. The movements of the rumen are infrequent, slow and sluggish, or are entirely absent. The same is true as to the rumen sounds.

Incompleteness or absence of the rumen movements or sounds does in itself not prove that the muscularis does not contract, or contracts less energetically. The movements of the rumen can become visible, and the sounds audible only when the contents of the rumen are propelled.

Defecation at first takes place at normal intervals; later an increasing constipation develops. There may exceptionally be diarrhea in dilatation of the rumen due to juicy feed (leaves of beets, beets).

Respiration is at first only moderately accelerated and superficial; as meteorism develops secondarily, it becomes more and more forced. The pulse beat is influenced in an identical manner. The secretion of the milk in cows becomes much diminished.

Occasionally weakness of the hind legs with staggering gait, inability to stand up, and even extension of the paretic weakness to the anterior part of the body or a picture similar to parturient paresis has been seen in cattle and sheep. (Schmidt, Pröger, Born).

Course. The disease may make its appearance either immediately after the ingestion of food, or several, up to twelve hours later, and then it slowly reaches its climax, the time elapsing after ingestion depending upon the nature of the particular food. After ingestion of easily fermentable feed, development may be as rapid as in acute meteorism. The disease usually *terminates in recovery*. The very mild cases frequently end in recovery on the first or second day and without any treatment whatever. In severe cases, even under the proper diet, the animals may be sick for three to ten days or even longer. Severe cases end only exceptionally in recovery in a short time, namely, when the contents of the rumen are diminished considerably in consequence of vomiting. Recovery is indicated by the return of appetite, rumination, and by the visible movements of the rumen, frequent belching and abundant defecation. If properly and timely treated, the disease rarely takes a fatal course, nevertheless many fatalities have been observed in sheep. Death may occur in consequence of severe meteorism in the second half of the first day of sickness; in sheep even with apoplectic rapidity. If the disease has been caused by the ingestion of dry or heavy food, or of food containing abundant flour or boiled potatoes, then the animals die only after the course of several days, or even some weeks, from inanition or gastro-intestinal inflammation, which is announced by elevation of temperature, rapid and weak pulse, clouding of the sensory organs and general weakness.

Diagnosis. The sudden appearance shortly after a rich meal, the enlargement of the abdomen, especially in the lower portion of the left side, the firm consistency of the rumen and considerable dulness on percussion in this region, are symptoms characteristic of the disease and distinguish it from the more fulminant development of acute meteorism. In the latter, atony of the fore-stomachs develops gradually, reveals an intermittent course without colicky pain and without stuffing of the rumen with masses of feed. Traumatic gastritis sometimes starts with symptoms similar to those of dilatation of the rumen, but it may be differentiated from the latter by the clinical history, the tenderness of the region of the xyphoid cartilage, the obstinate character of the disease and eventually by the administration of eseridine or veratrine in connection with arecoline (see foreign bodies in the fore-stomachs). Other inflammations of the gastro-intestinal tract run a febrile course in contrast to dilatation of the rumen and lack the filling of the rumen with doughy masses. Compression of the intestines, intussusception or torsion of the uterus are similar to dilatation of the rumen on superficial observation only, because the *ingestion of food and rumination cease suddenly and* there are colicky pains. However, a different history is obtained; there is no stuffing of the rumen and there is marked restlessness. Moreover, rectal and vaginal examination (spiral folds) show the distinguishing features.

Prognosis. *This depends upon the degree and duration of* the disease, but the nature of the ingested food must also be taken into consideration. The more fermentable the food or, on the contrary, the more dry it is, the longer the duration and the more unfavorable in general the prognosis. Complete recovery can, as a rule, not be looked for if the disease has existed six to ten days without any sign of improvement (Eisele). During a protracted course of the disease fatal complications arise frequently (gastro-intestinal inflammation or peritonitis), or chronic atony of the fore-stomachs remains as a sequel.

Treatment. Complete withdrawal of rough feed for one or two days is indicated before everything else, an improvised muzzle being used to prevent the ingestion of straw (Eber). Another important step is massage of the rumen to assist in or directly bring about the movements of the rumen. The effect of massage can be increased by methodical leading around of the animal and by repeated frictions of the regions of the flanks and sides.

Massage of the rumen is to be carried out by making moderate pressure with both fists along the whole left flank, both upward and downward, and also boring motions upon the contents of the rumen. Massage is kept up for five to ten minutes and, except in the case of sows near parturition, it is followed by the same procedure on the right side. When the rumen is very much overfilled, massage

must be carried out simultaneously on both sides by two persons. The procedure is repeated every two to three hours; in milder cases, however, only two to three times a day (Eber, Schlampp).

Cases of dilatation of the rumen of moderate severity may be cured by the withdrawal of rough feed and by a systematic massage. The latter can be supported effectively by certain laxatives and these should be administered, particularly in the severe cases. Best suited are those remedies which can be injected subcutaneously, for instance, eseridinum tartaricum (eseridine 0.2-0.3 gm., acid tart. 0.01 gm., aquæ dest. 20 cc. for adult cattle [Eber]), veratrinum sulfuricum (0.1-0.15 gm. for adult cattle; 0.03-0.04 gm. for goats and sheep, dissolved in fifty parts of alcohol), eserine (0.15-0.20 gm. for cattle [Feser]); the latter, however, sometimes causes excitment, dyspnea, abortion or even suffocation, and should not be used in small ruminants. Pilocarpine (0.3-0.5 gm.) may be used in cattle, without hesitation (Kunke). Likewise very serviceable is tartar emetic (for cattle 3 to 4 gm., for sheep and goats 0.3 to 0.5 gm. three times daily every 2 hours per os; great care in the administration), also barium chloride (cattle 6 to 10 gm. in 1 liter of water twice daily per os at the most), and less serviceable is veratrum (of the powdered rhizome for cattle 3 gm. three times daily; for sheep 1 to 2 gm. once daily; of the tincture the same amount or double the amount; for goats impractical [Scholl]). The action of tartar emetic and of veratrum is combined in drasticum "E. Barck," of which cattle should receive 75 cc. at a single dose, 6 months old calves 20 cc., and the administration should be repeated in case of necessity after 24 hours. The combined action of arecoline and veratrine is secured by means of arecovetrol or of arecoline-veratrine (Anti-paresin, Holterbach). Arecovetrol consists of eight numbered gelatine capsules with 0.1 gm. of hydrobromate of arecoline in the odd numbers, and 0.1 gm. of sulphate of veratrine in the even numbers with the addition of nux vomica. The eight capsules are given one after the other at intervals of two hours, with a bottle of linseed decoction; in case of need the course is repeated once. Arecoline-veratrine contains the drugs in the same quantities in solution and in similarly numbered vials. Its administration is the same. According to Schwab, istizine (adult cattle 30 to 50 gm., calves 2 to 5 gm., goats and sheep 3 to 5 gm.) acts a little more quickly (after 15 hours) than veratrum, but the administration of a combination of the two is more practical. (Istizine is excreted through the milk and when administered in large doses may cause diarrhea in the suckling young.) The internal administration of other medicines appears superfluous, except, perhaps, hydrochloric acid and bicarbonate of potash with equal parts of common salt; these may be given to stimulate the appetite after the removal of the masses from the rumen has begun (Eber).

Threatening suffocation can be successfully combated by rumenocentesis only in cases of rapid evolution of gases, while otherwise only the incision of the rumen with removal of part of the contents and in case of necessity irrigation of the rumen promises success. Following improvements or the incision of the rumen, the animals should under all circumstances be kept on a restricted diet and be given only good roots or other fresh food of good quality.

Incision of the rumen is performed on the standing animal in the region of the left flank by passing a pointed knife into the rumen and then making an incision 7 to 8 cm. long in a vertical direction through all layers of the abdominal wall. The wall of the rumen is then sutured by means of a glover's suture to the edges of the wound in the abdominal wall. The operation may also be performed by first making the incision only through the skin, whereupon the rumen is sutured to the abdominal wall in the form of an oval, and following this the incision into the rumen is completed within the oval space thus secured.

(b) Acute Bloating of Ruminants. Meteorismus acutus ruminantium.

(*Akutes Aufblaehen der Wiederkäuer, Trommelsucht, Blähsucht, Auflauf* [German]; *Tympanitis; Indigestion gazeuse ou méphitique, metéorisation aiguë* [French].)

Acute bloating of ruminants is a morbid condition with rapid dilatation of the rumen and reticulum in consequence of rapidly-forming gases.

Occurrence. Acute bloating of ruminants is seen frequently and particularly during the warm season and in animals on the pasture. Cattle and sheep are affected most commonly, goats more rarely. The disease appears often simultaneously in several or many animals of the same herd or of the same neighborhood.

Etiology. The cause of acute bloating is the abundant ingestion of easily fermenting food. Most dangerous in this respect are leguminosæ (clover, alfalfa, esparsette, vetches, peas, lentils, buckwheat) before they have flowered, i. e., at a time when they contain much water and are fine in texture. However, other green foods (young field plants, luxuriant grass, leaves of cabbage, beets, and of young potatoes; in sheep also cereals germinating in stubble fields); vegetable bulbs (beets and potatoes) also germinated malt, waste of sugar beets, draff, are liable to cause acute bloating if the animals have ingested large amounts of such substances without much dry food.

In sucking or weaned calves the disease sometimes occurs after the ingestion of unusually large amounts of milk.

While all these food stuffs may cause acute bloating, they vary considerably as to their dangerous nature. According to general opinion, clover, particularly red clover, is most dangerous; but even the same food material varies. The more luxu-

riant and the more juicy the food, the more easily it causes acute bloating. The greatest prevalence of acute bloating occurs in deeply-situated valleys with rich soil, and in years when the spring has been cold, and when the plants, which had been backward for some time, grow the more luxuriantly when warm weather sets in. The same circumstances cause bloating after pasturing on fresh stubble fields where weeds have been exposed to the sun a short time only and where dropped-off grains have germinated. The dangerous nature of feed plants becomes increased when they are strongly moist with dew or frosted or frozen or wet with rain. Bloating, therefore, is generally more common during pasturing in the early morning, during cool, moist weather, in spring and in fall. The danger is further increased by watering immediately after pasturing or after feeding. A withered green feed or green feed heated in consequence of storing it in large masses also causes bloating more easily than freshly-cut feed. This explains the frequent occurrence of bloating on holidays during stable feeding. Potatoes, beets and bulbous plants are more dangerous after decay has begun than when they are fed fresh. It is also claimed that bloating is easily brought about when animals swallow a lot of air while pasturing against the wind.

Predisposition to acute bloating varies as to the individual susceptibility. Greedy feeding predisposes to the affection, hence we see it frequently in starved animals and in animals permanently or generally stabled, when they are driven to a dangerous pasture. The variable efficiency of the muscularis of the rumen also plays a rôle. Physical debility, convalescence from various diseases during permanent stable feeding cause the movements of the rumen to be less energetic than under more favorable circumstances; the same occurs from violent motion or physical effort directly after the ingestion of food. Adhesions of the rumen may completely interfere with the movements of the organ.

Bloating occurs as an affection secondary to the ingestion of certain poisonous plants (spotted hemlock, water hemlock, species of ranunculus, colchicum autumnale, tobacco, mould). Obstruction of the esophagus causes severe meteorism, particularly if the food previously ingested is fermentable.

Pathogenesis. In consequence of the rapid formation of gas the rumen becomes dilated in a short time, and the disturbances are similar to those caused by overfilling of the rumen combined with meteorism (see page 82), but with this difference that bloating occurs much more rapidly and the symptoms are more fulminant, occasionally even leading to rupture of the rumen.

Anatomical Changes. A considerable increase of the abdomen and of the rumen becomes at once manifest. The wall of

the rumen is so tense that it can hardly be pressed in at all with the hand. Sometimes, one may be able to recognize during life a rupture of the rumen or of the diaphragm. On post-mortem the lungs are compressed by the diaphragm bulging into the thorax; they are dark red and full of blood. The vessels outside of the abdomen, especially the veins and particularly those of the subcutaneous connective tissue, are filled to their utmost with black red, possibly uncoagulated, blood. The signs of death from suffocation also include punctate and striate hemorrhages into the serous membranes, especially into the pleura and pericardium.

On post-mortem examination the rumen is found to contain soft, mashy, fermenting food masses and large amounts of gases which are inflammable and burn with a blue flame.

The composition of the gaseous contents of the rumen varies according to the type of food and to the nature of the fermentative process. Carbonic acid is always present to a large extent (50 to 80%); the amount is the larger the more fermentable the feed has been and the larger were the morsels upon ingestion. Next comes methan gas (CH_4) which makes up 16 to 39% of the gases. There are also present sulphuretted hydrogen, nitrogen and oxygen, the latter two derived from the air which has been swallowed. Oxygen, however, may be used up completely during the fermentative process. In starving animals the amount of carbonic acid becomes smaller, while methan, nitrogen and oxygen are increased (Lungwitz).

Symptoms. The animals cease to feed, stand motionless, place their feet under the abdomen or spread them, curve their back, turn their head around towards the abdomen.

A short time later an increase in the circumference of the abdomen becomes noticeable; it soon reaches a high degree. The increase takes place all around the abdomen, becoming, however, most marked on the left side, particularly before death. The flanks, especially the left one, become flattened; in severe cases the left flank may so protrude that its apex is higher than the back (Fig. 7). The abdominal walls are very tense, can be pressed in only with difficulty, and at once protrude again after removal of the pressure. The left hand placed over the region of the rumen cannot feel any movement of the latter. Percussion over a large territory, sometimes down to the lower third of the abdomen, is of low pitch, excessively full, not tympanitic and occasionally metallic, in the region of the left flank. As bloating progresses, and as the walls of the abdomen and of the rumen become more tense, the percussiou sound becomes weaker, without, however, becoming very weak. The rumeu sounds (friction) are no longer heard; crepitation, however, becomes more distinct.

Occasionally gases are expelled from the rumen and vomiting may exceptionally occur.

As the abdomen enlarges, respiration becomes more and more difficult. The animals attempt to move especially the mid-

dle ribs to a higher degree, they dilate their nostrils, open the mouth and stretch out the tongue.

The mucous membranes soon assume a bluish-red color, their vessels become dilated and form a well-marked reticulum, particularly in the conjunctivæ; the veins of the subcutaneous connective tissue (on the neck, on the udder) likewise become dilated. The pulse is accelerated in proportion as the abdomen is more and more distended; the pulse then becomes gradually weaker and finally can no longer be felt.

In the further course of the affection the animals move *restlessly about, expressing intense anxiety, and perspiration* appears on the skin. Foamy saliva flows from the mouth. The region of the back and croup sometimes develops a cutaneous emphysema (Reynal, Woehner).

Fig. 7. Meteorismus acutus in a goat.

In very severe cases the general cyanosis gradually increases, the heart beat becomes bounding, the extremities and the ears are cold, the animals stagger, break down and finally die with convulsions.

Course. After the ingestion of large masses of very rapidly fermenting feed, gas is formed very quickly, even during

digestion or immediately after it, and the gas formation progresses so rapidly that suffocation may occur within a few hours. In other cases the development of gas is slower, powerful convulsive contractions of the rumen, and frequent belching in combination with vomiting preventing the excessive accumulation of gas; in the meantime the fermentation itself becomes less and the animals recover completely.

Diagnosis. Rapid increase in the size of the abdomen in connection with strong tension of the abdominal wall, increase in the intensity of the percussion sounds over a large area, give *a characteristic clinical picture. The character of the sound on* percussion and the changes in the left flank distinguish the disease from overfilling (dilatation) of the rumen; chronic bloating and atony of the fore-stomachs are much slower in development and their symptoms occur in frequently repeated attacks. Bloating due to obstruction of the esophagus or of the cardia is diagnosticated on the basis of a different history and in the former the sound encounters an impediment; belching is also absent. It must not be forgotten that gastritis due to foreign bodies sometimes is initiated by acute bloating.

Prognosis. Acute bloating is usually much more dangerous in sheep than in cattle, evidently for the reason that in the comparatively small body of the sheep a minor excess in gases may bring about serious disturbances of respiration and circulation and that the comparatively weak muscularis of the rumen is unable to overcome the pressure and expel the gases. The prognosis (aside from those measures which may bring about recovery) is the more unfavorable the larger the amount of ingested food and the more fermentable it is. Everything else being equal, bloating caused by clover is most dangerous, because a fulminant course brings about an intimate admixture of the food contents and the gases. The course of the affection is also unfavorably influenced by debility, early youth or advanced age and preceding disturbances of nutrition. The more animals get sick simultaneously, the more losses are to be expected, because the smaller is the possibility of timely treatment.

Treatment. Massage of the flank must be practiced on both flanks while the foreparts are elevated (page 84); according to Eber, this procedure is the most reliable and simplest method to remove bloating in cattle, *except when bloating has been produced by obstruction of the esophagus.* Compressing the abdomen *by tying a rope around it or a constant, strong, even* pressure on the left flank, may also bring about good results (Rychner).

Elevating the animal serves the purpose to bring the gastric entrance of the esophagus in a position above the gases over the masses of feed, and this may be accomplished in the following manner: A stable floor or a strong board is laid obliquely over a box, barrel or bundle of straw. The animal is then placed

with its front legs on the improvised inclined plane and supported in this position by two persons who get a hold of the thorax. In an emergency the front feet of the animal may be placed in the crib (Dommerheld). Some kind of an arrangement also must be improvised if the animal is on pasture. Smaller animals like sheep may be placed on their hind legs.

The effect of driving up hill is similar to that of massage of the rumen with the animal raised in its foreparts, and it is, therefore, often made use of with good results in mountainous countries (Eber). Energetic contractions of the rumen may also be produced by freely soaking the flanks with cold water, or by driving the animals into a river (the latter method is to be recommended if numerous cases of the affection occur simultaneously in sheep).

The effect of massage of the rumen is materially assisted by such measures as will cause belching or vomiting. A very simple and frequently effective method consists in irritating the velum and the pillars of the fauces of the animal with a dull, flexible rod (the blunt end of a whip wrapped with cloth); while this is being done the mouth must be forced open and the tongue drawn out. This frequently causes a good deal of belching with the expulsion of very large amounts of gas, sometimes mixed with food particles. It is advisable to stand on the side of the head of the animal, so that the very fetid gases do not get directly into the face of the practitioner. It is less satisfactory to merely pull out the tongue of the animal, moving it up and down in the mouth with a rope provided with knots or made of straw, or to apply a straw rope soaked in tar or other nauseating substances to the mouth of the animal.

The removal of the gases in the rumen through an artificial opening is only indicated if the condition of the patient becomes worse in spite of all other means, if suffocation is threatening, or when the animals are on the floor, so that the common methods of treatment have become impossible. The introduction of the stomach tube is not a very dangerous procedure; but it is usually not successful in primary bloating, since the inner opening of the esophagus lies behind the masses of food, so that the tube will immediately become obstructed by food particles. This defect may be remedied by an elevation of the foreparts of the animal; however, when this is done, massage is usually sufficient and makes the use of the stomach tube superfluous (Eber). If the dyspnea is severe, the introduction of the stomach tube may be injurious and may lead to fatal asphyxiation.

Puncture of the rumen must be reserved for cases of primary meteorism after the other methods have failed and when there is immediate danger of death. In some cases puncture of the rumen, particularly if performed by laymen, may cause a more or less extensive peritonitis or the adhesion of the rumen to the abdominal wall, which will subsequently endanger the health of the animal, more or less.

Puncture of the rumen is performed with a long trochar held in the closed hand; it is pushed (contained in its protecting tube) into the most prominent point of the left flank or into the center of a line which connects the external angle of the ileum with the middle portion of the last rib. An incision into the skin may have been made previously. The point of the trochar is directed towards the right elbow. After penetration the stiletto is withdrawn and it or a thin rod is used to remove particles of food which may collect in the tube of the trochar. In order to prevent too sudden congestion of the abdominal vessels or rupture of vessels, or anemia of the brain, it is advisable to let the gases escape gradually. The tube of the trochar should then be closed with a cork and left in place for several hours, safely secured by a bandage wound around the rump of the animal.

In an emergency, puncture of the rumen may be performed with a strong pointed knife. The latter, first carefully cleaned, is held with its blade towards the front and is pushed 8 to 10 cm. deep into the rumen; it is then rotated at a right angle in order to produce a gaping wound.

The punctured wound usually heals rapidly, but a wound which has been made with the knife should be closed by two or three sutures.

The internal administration of drugs plays a very subordinate rôle in the treatment of primary meteorism and even so-called neutralizing agents (ammonia, lime water, potash) will not materially diminish the amount of gases contained in the rumen. However, the subcutaneous injection of medicines which stimulate the movements of the rumen may in some cases be indicated (see page 85). A moderately advantageous effect might also be expected from agencies liable to inhibit fermentation. Among these may be mentioned subsulphate of sodium (100.0-200.0 or 25-30 gm.), chlorate of potash (50.0-60.0 or 5-10 gm.), resorcin (15.0-20.0 or 5-8.0 gm.), hydrochloric acid (one tablespoonful to a quart of water) or lime water (1-2 to 1/4 or 1/2 quart). Tar preparations must be avoided because they impart a disagreeable taste to the meat and would make it unpalatable in case slaughtering of the animal becomes necessary. The medicines mentioned above are to be given in moderate dilutions; however, in bloating of a high degree their administration meets with considerable difficulty, because the high pressure present in the rumen prevents deglutition and the restlessness of the patients causes danger of aspiration. The medicines may also be introduced through the stomach tube or, after puncture of the rumen, through the tube of the trochar.

After the subsidence of acute symptoms, the animals should be subjected for several days to a strict diet, so that all easily fermenting material is removed from the rumen.

Moderate doses of salts or medicines stimulating the removal of the contents of the rumen are indicated. In secondary bloating, particularly if due to obliteration of the esophagus, puncture of the rumen cannot be avoided, in case the esophageal obstruction cannot be removed speedily.

Prophylaxis. Primary acute bloating is always due to careless pasturing or improper feeding and can be prevented with care and attention. It is necessary to educate shepherds and attendants by proper instruction. Since leguminosæ are

most dangerous, it is proper not to pasture ruminants on such pastures, and the restraint should include barley stubble fields. Animals should be permitted to run on such fields only after they have received some dry feed or have fed on less luxuriant pastures, so that they will not take up too much of such juicy vegetable feed. They should never be left long in dangerous pastures and should be moved about during pasturing. The same precautions should be observed in feeding on very luxuriant pastures and particularly on dewy mornings or after rains.

Freshly-cut green feed is to be given in smaller rations and mixed with larger amounts of dry feed the younger and the more juicy such plants are. The same precautionary measures must be observed when feeding easily fermentable material (bulbous plants, floury feed, germinated malt, etc.). One should always avoid feeding material which is already fermenting or heated; if in this condition it should be cooled, dried aud mixed abundantly with dry feed. The care-takers of animals should be made familiar with the simplest methods of expelling gas from the rumen. Friedberger & Fröhner recommend that farms and communities with large herds should always have trochars in stock.

(c) Chronic Bloating of Ruminants. Tympanitis chronica ruminantium.

Chronic bloating of ruminants is only a group of symptoms which may be present in a variety of diseases of the fore-stomachs, the abomasum, the esophagus and the intestines.

Etiology. Chronic tympanitis is observed in diseases in which the removal of gases from the rumen, either by belching or by the intestinal tract, is prevented for a considerable time. The amount of gas formed may be normal or the gas formation may be more lively than normal, without, however, reaching a high degree. Chronic bloating occurs preferably in atony of the fore-stomachs, both primary and secondary.

Another frequent cause is traumatic gastritis, while in calves and occasionally in lambs also, bloating is due to the occlusion of the openings of the fore-stomachs or of the pylorus by hair or feed balls. Disease of the abomasum likewise occasionally produces chronic tympanitis.

Another cause is stenosis of the esophagus (see page 59) which preveuts the removal of rumen gases by belching, and also rumination. Stenosis of the esophagus is most commonly produced by tuberculous mediastinal lymph glands as pointed out by Johne. Weber and Goldmann have each reported a case of compression of the lower end of the esophagus by large echinococcus cysts of the liver. In a case of Ade, the lower end of the esophagus was compressed by the reticulum which had entered the thoracic cavity through a slit in the esophagus.

Esophagus dilatation may also be the cause of chronic tympanitis, because the former leads to occasional temporary obstruction of the esophagus.

Stenosis of the intestines may likewise be the cause of chronic tympanitis.

Anatomical Changes. Aside from the fundamental affection there is intensely fermenting food in the rumen, as well as large amounts of gases. The gases contain less carbon-dioxide than in acute bloating. In a case investigated by Erdmann, he found 42% CH_4, 32% CO_2, 20% N. and traces of hydrogen. Lungwitz found the proportion of hydrocarbons to carbon-dioxide as 1.1 :1.2-1.5.

Symptoms. Chronic tympanitis consists in a gradually increasing, generally moderate, bloating of the left flank depression; it occurs at first at irregular intervals, later, however, and in profound cases, regularly after each feeding. It may exceptionally be present as a permanent condition. The degree of bloating is influenced by the character of the food; if the latter is very fermentable, bloating is more noticeable and may then be very extensive. The movements of the rumen may be perfectly normal or, in severe cases, they are somewhat diminished or decidedly sluggish. Rumination may be more or less suppressed or completely abolished. In the latter case other gastro-intestinal disturbances become manifest.

Eber wants the term chronic tympanitis reserved for those morbid conditions where the function of the stomach is not disturbed at all or shows a very minor degree of disturbance in spite of bloating of a high degree.

Course. Chronic tympanitis usually develops slowly and extends over weeks and months. The continued pressure of the enlarged rumen upon the left transverse processes of the lumbar vertebrae gradually produces a slight rotation of these vertebrae on the longitudinal axis. This symptom may be of importance for determining the duration of the underlying disease (tuberculosis of the mediastinal lymph nodes) (Johne). Its course is determined by the underlying primary affection.

Diagnosis. The periodic appearance of generally moderate bloating is sufficient for a diagnosis. Since chronic tympanitis is only a symptom it is always desirable to diagnosticate the underlying primary affection. It is then important to remember that chronic tympanitis due to an affection of the esophagus or depending upon tuberculous mediastinal glands is characterized by periodically recurring attacks of bloating of the rumen without disturbance of the gastric fuuction or in very severe cases with little disturbance; also that all symptoms cease after the removal of the gases through the esophageal tube or through puncture of the rumen and in passing the stomach tube a certain resistance will be felt when the end of the tube reaches

the middle part of the chest cavity. In chronic bloating due to intestinal stenosis the gastric function is disturbed and disturbances of defecation are particularly well marked.

When other gastric disturbances are associated with chronic tympanitis, primary or secondary disease of the stomachs must be looked for (atony of the fore-stomachs, foreign bodies).

Prognosis. Chronic tympanitis does not endanger life *per se;* however, its periodic appearance disturbs the nutrition to a certain degree, but the emaciation which finally comes on is really due to the underlying primary disease. The latter also determines the prognosis.

Treatment. The removal of the fermenting food may be accomplished by stimulation of the movements of the rumen through massage of the rumen in an elevated position and by the administration of certain medicines, mentioned before (see pages 90 and 91). In addition laxative salts may be given (200.0-300.0 gm. or 10-25.0 gm. for smaller animals) combined with tartar emetic (2.0-3.0 gm. or 0.3-0.5 gm.), aloes (5-10.0 gm. or 2-3.0 gm.) or ipecacuanha (4-6.0 gm.). Such medicines must be given repeatedly. Anti-fermentative solutions (see page 92) are also useful, particularly disinfectants, and eventually also lavage of the rumen (see page 105). After the morbid condition has been removed, one should administer, with proper feeding, some more laxative salts, but in smaller doses (30-50.0 gm. or 5.0-10.0 gm.). Good effects are also seen from chloride of barium (6-8.0 gm.) dissolved in water (Faber, Marder).

In the presence of an incurable underlying condition, it is advisable to slaughter the animals. If there is reason for suspecting a foreign body in the stomach, incision into the rumen may bring about recovery; this procedure should particularly be considered in the periodic tympanitis of calves.

(d) Atony of the Fore-stomachs. Atonia ruminis, reticuli et omasi.

(*Chronische Unverdaulichkeit, Chronische Indigestion, Chronische Dyspepsie, Chronischer Magendarmkatarrh des Rindes* [Friedberger & Fröhner]; *Pansenlähmung* [Dieckerhoff]; *Ermüdung [Parese] des Verdaungskanals* [Pütz]; *Löserverstopfung, Sporadische Löserdürre, Chronische Entzündung des Lösers* [Anacker] [German]; *Indigestion chronique des ruminants, Obstruction ou Engouement ou Indigestion du feuillet, Omasite ou Gastrite chronique, Météorisation chronique* [French].)

Atony of the fore-stomachs consists in a slowly developing weakness of contractions of these organs, depending upon a

diminution of irritability and energy of contraction. The removal of the normal or even subnormal amount of food becomes more or less retarded and digestion disturbed.

Occurrence. The disease is most commonly observed in cattle, somewhat less frequently in goats, occasionally in sheep. The frequency of its occurrence depends upon the prevailing conditions of feeding. The disease is frequently met among animals of poor people, in the neighborhood of larger cities, or in such parts where milch cows and cattle in general are fed permanently with various substitutes for natural feed or with relaxing or, on the contrary, with irritating feed. Most cases occur during the colder season, especially in spring, because then an irrational method of feeding is mostly resorted to. *During certain seasons or in districts with poor crops the disease* is particularly common. Finally, the disease is seen in some parts in very young calves.

Etiology. As a **primary** acute affection we find atony of the muscularis of the first three segments of the stomach after *a sudden change of food* (after change of owner), as a consequence of overexertion during long transit, or after difficult parturition. An insufficient function of the muscularis of the fore-stomachs also occurs after intense psychic stimuli (excitement, pain, longing for the calf). Atony due to these causes is probably combined with lack of secretion in the abomasum and usually disappears in a short time, but if neglected it may lead to the more serious chronic atonic condition. The same may be said of the acute atony seen after the course of acute, febrile affections or after parturient paresis which may last for some time and which may be intensified by the aspiration of the contents of the reticulum.

In its usual chronic form atony of the fore-stomachs may occasionally be caused by dilatation of the rumen, particularly if this affection takes a protracted course, in consequence of which the muscle fibers become paretic.

Usually, however, the affection is due to prolonged improper feeding. As harmful in this respect must be mentioned food which is dry, not easily digestible and not very nutritious, such as chaff, straw, marsh hay, garbage from the garden or the kitchen, particularly if it contains fragments of bones, also oil cakes, bran, shelled grain or leguminosæ. Not less dangerous are tough, fibrous food stuffs, such as coarse alfalfa, raspberry bushes, brush hay. Snodgrass saw several cases together in cattle on pasture on which the grass was dead or wet. Their obnoxious effect is due to the fact that many of them are easily converted into densely matted masses that are hard to move, and especially to the fact that they no longer

contain protein or starch-splitting vegetable enzymes, or at the most but very small amounts of them, or have lost the power of fermentation by destruction of the embryos in the seed through the milling processes and thus are no longer able to furnish products of decomposition that would excite movements of the fore-stomachs.

The disease is liable, on the other hand, to make its appearance if the animals have been fed for a long time with fermentive, spoiled, wet or frozen feed; also with boiled potatoes or with a thin floury mash (Eberhardt) without any rough feed at all.

According to J. Schmidt, disturbances in the activity of the fore-stomachs together with certain symptoms of intoxication are observed after feeding excessive amounts of beet roots (Runkelrueben), less frequently from rutabaga (daily 45 kg. and more to lactating and 20 to 25 kg. to nonpregnant and pregnant cows). Especially dangerous in this respect, and more so in the last months of winter, are sprouted, spongy and partly decayed turnips as well as turnips fed immediately from the turnip pile.

Other causes that may be enumerated are insufficient mastication and mixing of the food with saliva, in consequence of faulty teeth or in chronic disease of the tongue, greedy feeding, or the frequent prevention of rumination in work cattle.

The affection develops in young calves in connection with gastro-intestinal catarrh, if these animals, after having previously been fed exclusively with milk, receive too suddenly or too early, bread, crushed oats, germinating malt, hay, etc., in addition to skimmed milk (Imminger).

Predisposing causes are early youth and old age, and debility after chronic, acute and febrile diseases.

Secondary atony of the fore-stomachs cannot usually be differentiated clinically from the primary form. An exception is that form of secondary atony which is observed in the course of traumatic gastritis, and other forms of inflammation of the fore-stomachs or of the abomasum. For this reason and in consideration of etiologic factors, it will be treated in a special chapter (q. v.).

In the advanced stages of pregnancy all sections of the stomach are sometimes so compressed by the pregnant uterus that atony results. More extensive adhesions of the fore-stomachs among each other, or with neighboring organs, diminish the functional activity of the fore-stomachs, as does also prolapse of the omasum into the thoracic cavity. In the case reported by Richter the atony was due to a dislocation of the gravid uterus downward and toward the left side and a dislocation of the posterior end of the rumen toward the right flank. Other causes are tumors (see tumors of the stomach), diffuse lymphadenitis of the wall of the fore-stomachs or of the aboma-

sum (Eber), certain diseases of the abomasum (obstruction by foreign bodies or feed material, tumors, ulcerations), subacute or chronic peritonitis, even if it has not caused adhesions of the fore-stomachs, chronic disease of the liver (chronic interstitial inflammation, echinococcus, tumors, gallstones), certain chronic diseases of the lungs and heart.

Pathogenesis. In consequence of the enumerated causal conditions, especially, however, in consequence of continued improper feeding, there occurs a diminution of the irritability or of the energy of contraction of the fore-stomachs, or both functions of the latter become diminished. The resulting weaker and less frequent contractions of the fore-stomachs are the cause of a deficient mixing and of a retarded removal of the contents. According to prevailing conditions and amounts of the feed contents, the degree of disturbance of function and the amount of ingested water, there occurs either a lively formation of gas or the contents desiccate more or less; even in the latter case formation of gas is rarely absent. Every additional act of ingestion of improper food further increases the disturbances.

No matter in what section the primary disturbance, desiccation of the feed always occurs in the rumen and omasum, that is, in those sections where it is even normally less rich in water than in the reticulum where the contents rarely desiccate.

The reason for this behavior lies in the functional inter-relation of the individual sections of the stomach; stoppage of movements in one section also causes stoppage of movements in the other sections. It can not be denied, however, that occasionally the omasum alone is the seat of the obstruction. This occurrence is plausible since normally the contents of the omasum are comparatively firm, its muscularis weak, and since finely divided feed (short cut chaff, bran, etc.) may occasionally get directly into the third section of the stomach without rumination. *These are however rare cases and their differentiation from other forms of atony* appears impossible since disease of the omasum at once also leads to disturbance *of the functions of the other sections of the stomach.*

Incomplete mixing and accumulation of feed masses lead to disturbances of digestion and to lack of nutrition, and these again further increase the atony. Abnormal fermentation of feed in the fore-stomachs may form poisonous decomposition products, while the continued or often repeated dilatation leads to a permanent dilatation of the fore-stomachs. If the affection has lasted a long time, there also result inflammatory or even necrotic changes of these sections. For these reasons catarrh of the abomasum and of the small intestines is one of the common sequelæ and the deeper inflammatory processes may spread from the walls of the fore-stomachs into the peritoneum.

Anatomical Changes. Rumen and omasum occasionally also the reticulum, are more or less dilated. The increase in size of the omasum is particularly obvious, although in young calves the rumen is most enormously dilated (Imminger). The con

tents of the omasum are unusually dry, almost rocklike, but they can be ground up into a fine powder between the fingers; they completely fill the omasum (*"Löserverstopfung"* [German]). The rumen likewise usually contains desiccated, fetid masses of feed and usually also gases. If the affection has been caused by feeding thin mashed, or boiled potatoes, the contents of the rumen look like street mud or like a thin dough (Sipp, Eberhardt), while the omasum may not be obstructed, as is occasionally the case in other cases of atony.

Desiccation of the feed-mash in the omasum without dilatation of the organ is a common but negligible finding in severe febrile general diseases or other affections in which the movements of the fore-stomachs have been diminished for a considerable time.

The mucosa of the fore-stomachs either does not show any changes at all, or it appears reddened uniformly or in spots, occasionally with hemorrhagic spots, if the process has lasted for some time. These changes can easily be seen postmortem, since the cornified squamous epithelia soon after death become detached in shreds or adhere to the masses of feed (common postmortem appearance!). One occasionally finds pressure-necrosis or hemorrhagic ulcers in the omasum, usually combined with a general acute peritonitis. The latter, however, may be present without pressure-necrosis of the wall of the omasum.

In secondary atony the causative underlying disease can generally be ascertained on postmortem examination.

Symptoms. In the **acute form** of the disease one only notices a diminution of the appetite and thirst; rumination is infrequent and sluggish and there is a decrease in the energy of the rumen movements which are, however, normal in frequency (simple indigestion [Eber]). If these symptoms, which are not very prominent, are overlooked, and if the animals are not properly treated, the clinical picture of the chronic form gradually develops.

The **chronic form** usually develops very gradually, although it may occasionally evolve within a very few days into a characteristic, clinical picture. The animals from time to time take less feed and they generally prefer rough feed; in some cases the appetite may be perfectly normal for several hours and even for a few days. Thirst is generally diminished, but it may also be increased (Bouley, Heu). Here and there the animals lick greedily. Rumination is, as a rule, gradually diminished and it may also occasionally become suppressed suddenly. As a rule rumination only occurs from time to time; after a longer interval the masticatory movements are sluggish and reduced in number. Belching occurs rarely and it expels fetid gases, becoming more frequent after more pronounced meteorism. Gagging or vomiting is rare; if the latter does occur, it expels a thin-mashy, fetid material.

The circumference of the abdomen is frequently increased on the left side and below, with bloating of the left flank, which may even become quite prominent; if, however, there is a continuous lack of appetite, the circumference of the abdomen may be diminished. The rumen is usually larger, even in deficient digestion of food, sometimes only moderately filled or almost empty; it generally feels hard, rarely fluctuating. The upper part of the rumen is filled with gases, so that the consistency of the solid contents of the rumen can only be ascertained by strong pressure upon the left flank or by rectal palpation, or in smaller animals by simultaneous palpation on both sides. Bloating of the rumen usually remains within moderate limits; it is, however, subject to considerable variations because it increases more or less after the ingestion of feed, diminishing again or even entirely disappearing (chronic meteorism). Every improvement of the appetite with an increased ingestion of feed promptly leads to an increase of meteorism which may become permanent if the gastric movements cease entirely. The movements of the rumen at first are diminished inconsiderably in frequency and energy, so that the disturbance may easily be overlooked; without proper treatment the movements of the rumen may cease entirely after a few days. Disturbances of the movements of the rumen are subject to similar variations as meteorism; pressure upon the rumen sometimes elicits pain.

An increased resistance may, according to Detroye, be felt in the abdominal cavity in obstruction of the omasum if the fist suddenly makes firm pressure behind the right costal arch; the animals simultaneously emit a dull groan. Hink states that the reticulum is tender to pressure when percussion is made over the lower posterior portion of the thorax, or to pressure upon the ensiform cartilage.

The rumen and intestinal sounds are suppressed, the latter, however, are loud and rumbling before the appearance of diarrhea. Defecation is less frequent and is finally obstinately suppressed. The feces are peatlike, blackish, their surface shining and they contain poorly digested food particles. Exceptionally, that is if the disease has been caused by feeding of mashy substances, no disturbances of defecation may be noticeable. Sooner or later constipation yields to malodorous diarrhea which may be permanent or may alternate with constipation. Sometimes mild colics supervene, particularly before diarrhea sets in.

The temperature remains below 40° C. as long as there are no complications. The pulse is at first normal as to frequency and strength, but becomes rapid and weak later on. The respiration is more or less accelerated, particularly if bloating exists, and expiration is accompanied by groaning and sighing, particularly on lying down. There also occurs not uncommonly grating of the teeth. In one case Petersen observed a dry cough after the disease had existed eight days.

The sensorium frequently does not manifest any disturbance for a long time, even if emaciation is already present, ex-

cept perhaps that the animals are rather sluggish and lie down a good deal. Sometimes the patients are somewhat excited and show twitching (Petersen).

In the further course of the disease or with complete lack of appetite, the general debility becomes more and more marked and the gait becomes staggering. Exceptionally one observes in male animals, also in cows, paretic conditions similar to the prostration of cows (Wucher) or to parturient paresis (Sticker, Schell, Eber).

In the further course, emaciation and weakness become more and more manifest, the eyes sink in, the hair becomes harsh, the skin dry and the dental pad, which is dry from the start, becomes furrowed with clefts. The secretion of milk decreases and finally stops entirely.

In secondary atony we are sometimes able to recognize symptoms due to the underlying fundamental condition.

The **poisoning by turnips** in cattle is manifested in milder cases by diarrhea, diminished appetite and decrease in the milk flow; in severe cases by stiffness of the joints and weakness in the posterior part of the body with slight swaying while in motion, during which animals will have to be pushed forward step by step. Only seldom an edematous swelling of all four legs or excessive flow of saliva is observed. In more advanced cases convulsions of the muscles of the head or of other parts of the body are observed and can be produced artificially by any excitement. In animals taken sick soon after calving general twitching of the muscles can be seen, and between such spells a condition simulating parturient paresis may be observed, which usually terminates fatally.

In the disease termed "acute licking disease of cows," which otherwise occurs under the same conditions, van der Veen observed symptoms of salivation and licking; the animals would not swallow the food taken into the mouth, would lick all objects within reach and would firmly press the lips against them and suck. Later the pulse was accelerated, the animals seemed excited, pressed the head against the thoracic wall, would finally fall down and could not get up, but recovered nevertheless.

In **young calves** the disturbances appear a few days after improper feeding (Imminger). The first symptom is violent diarrhea which may last for eight to ten days and may then stop suddenly. The fluid feces then become firm and gray and have an intensely fetid odor. As soon as scouring stops, the general condition is much disturbed and the previously good appetite is suppressed. Then chronic meteorism appears either very gradually or on the contrary very rapidly, and it is so intense that the rumen becomes much dilated and presses upon the rectum (Bitard). As a rule bloating occurs after each feeding and lasts six to twenty-four hours. As bloating occurs the animals become listless and curve their back. An elevation of temperature also occurs. After some time considerable emaciation is observed.

Course. Aside from acute cases which under proper treatment and diet end in recovery almost without exception, atony which has become chronic has a very variable course, and temporary improvements alternate with aggravation at variable intervals.

Permanent improvement may be looked for if simultaneously with the appearance of flat, grumous masses of feces, sup-

posedly coming from the reticulum, the bloating disappears, the rumen does no longer contain firm masses, its circumference becomes diminished, its motions become more frequent and more energetic, and the groaning and sighing subside. In adult animals improvement is very gradual and leads to complete recovery only after four to twenty-five days (Cadéac); however, it may be interrupted by a recurrence by improper attendance on the part of the owner of the animal. Atony due to compression by the uterus disappears within a few days after calving, in favorable cases. If properly treated, calves likewise recover within a few days (Imminger).

Unfavorable signs are entire absence of appetite and of the motions of the rumen, continuous bloating, constipation resisting every method of treatment or, on the contrary, continued diarrhea, fever or subnormal temperature, marked tenderness on pressure of the rumen, continuous sighing, inability to rise, a paretic condition, convulsions, cachexia.

The duration of the disease is from several weeks to several months (after Bouley twenty-five to thirty days, after Cadéac one to two months, after Dieckerhoff usually many months, even one to two years). Sometimes, however, the affection only lasts eight to ten days (seen by Eber in cows in advanced pregnancy).

Diagnosis. The clinical symptoms and the frequently helpful history of the case usually permit a diagnosis of atony of the fore-stomachs. Severe general diseases which in their course also lead to a diminution of the movements of the fore-stomachs, are from the start distinguished by fever and other prominent symptoms and also by their rapid onset. *Dilatation of the rumen occurs after excessive feeding of animals which were previously healthy. The rumen is considerably* dilated; if the condition is at all severe, its movement cannot be seen at all. Acute bloating could only be confounded with those cases of atony of the stomach where there has accidentally been a higher degree of meteorism. The rapid development of bloating following the ingestion of easily fermenting food in large amounts by a previously healthy animal, and the complete recovery in the shortest time after proper treatment, easily secure the diagnosis of acute meteorism. The forms of chronic bloating not depending upon disease of the fore-stomachs or abomasum (see page 94) are characterized by the fact that a collection of gas is the most prominent or even the only symptom, while the function of the fore-stomachs suffers only during strong bloating; besides all symptoms disappear for the time being as soon as the meteorism has been relieved. Acute gastro-intestinal catarrh and gastro-intestinal inflammation can be differentiated by their usually sudden onset, by fever, by the depression in the left flank, and the absence of gas and masses of feed in the rumen; in gastro-intestinal inflammation, there

are from the start signs of grave general disturbance present. If, however, acute gastro-intestinal catarrh or gastro-intestinal inflammation have developed in consequence of atony of the fore-stomachs, then the history only can clear up the case.

It is usually easy to decide whether primary or secondary atony of the fore-stomachs is present if one considers the history of the case, the condition of the other organs, and also the manner in which the affection is influenced by treatment. Gastric atony may be assumed to be due to compression by the pregnant uterus, when towards the end of gestation disturbance of digestion develops gradually without any errors of diet and when the uterus is unusually large. Prolapse of the omasum into the thorax produces a dull or tympanitic sound upon percussion of the lower third of the thorax on one or on both sides, while in place of the absent respiratory sound, strong peristaltic movements may be heard (Harms); there are also occasionally circulatory disturbances such as dilation of the jugular vein, diminution of the apex sound on one side (Liénaux); these are due to compression of the heart. In cases of secondary gastric atony one may observe the symptoms of chronic disease of the liver, lungs or heart or those of subacute or chronic peritonitis. However, frequently the seat of the primary affection cannot be recognized in the living animal.

The early recognition of traumatic gastritis which is indeed only a special form of secondary gastric atony is very important. Aside from the tenderness to pressure of the region of the omasum which is frequently present, and the sudden onset of obstinate gastric disturbances without any apparent cause, which will resist any and every treatment, the appearance of spontaneous pains after drugs which stimulate gastric movements (see page 120) point to a traumatic gastritis. Sometimes only the further course brings enlightenment, especially the subsequent affection of the pericardium, the pleuræ, the lungs and the subcutaneous connective tissue in the sternal region.

Prognosis. This depends primarily upon whether a primary or a secondary atony exists, upon the duration of the disease and upon the general nutrition of the animal. Primary gastric atony can generally be cured by timely treatment and proper regulation of diet. The prognosis of secondary atony depends upou whether the underlying primary disease is curable or not. Atony due to gestation usually disappears a few days after parturition, but even in this form death may occasionally occur (Eber, Frasch).

Aside from the last mentioned form of gastric atony, recovery cannot be looked for after the disease has lasted for two to three weeks and if the gastric disturbances do not yield to prolonged treatment, if constipation has existed several

days or scouring several weeks, if a paretic condition has set in or if symptoms of gastro-intestinal inflammation or of peritonitis make their appearance. Then the animals should be slaughtered. In tuberculous cows or in animals advanced in gestation the prognosis ought to be guarded even in primary atony (Eber).

Treatment. After withholding rough feed for one or more days, one feeds for a few days only good hay in small quantities and some floury fluids mixed with some common salt, gruels from grain or milk, in cases of atony produced by watery or cooked feed only small quantities of tender hay given repeatedly should be allowed, since feeding till the animal is satiated and immediately after the return of the appetite may be followed by aggravation of the condition. Pasturing has a good dietetic effect. Calves do best on boiled milk or boiled oatmeal, which should be given in small quantities several times a day (Imminger).

The treatment proper consists preferably in massage of the rumen (see page 85). Drugs stimulating motion of the rumen (see page 120) are likewise very serviceable and are eventually useful for a long time. According to the results of Gmeiner and his school, the motions of the rumen may be influenced favorably by alcohol which is given in fractional doses every two hours, in the form of spirits or of arak (cattle, 50 gm., goats 20 gm., per dose; arak should not be given to sheep), also claret (cattle, 150-250 gm., goats 50-100 gm., sheep 50-150 gm. per dose). Corn whisky or cherry spirits (100 gm., 50 gm., 50 gm.) rum (50 gm., 20 gm., 20 gm.). Bissauge used nux vomica (3 to 4 gm. daily) with good results. Purgatives should be given only in cases of constipation, since as a rule they have no effect upon the movements of the rumen and their continued administration with water only aggravates the disturbance of activity already existing. Diarrhea of somewhat longer standing ought to be combated by the administration of styptics.

A drug which has proved beneficial is hydrochloric acid (one to two tablespoonfuls in a quart of water several times daily); it does not only stimulate the digestive processes, but also hinders to a certain degree the abnormal decomposition in the stomach. This is also accomplished by creolin (5 gm. three times daily in a bottle of water) which has been recommended by several observers. The following drugs are also to be mentioned in this connection: Common salt and sodium bicarbonate āā; sulphate of sodium; artificial Carlsbad salt (one to two tablespoonfnls).

Cases which have not done well under any other mode of treatment have sometimes been benefited by the administration of large amounts of water directly into the rumen through a trochar introduced through the left flank and connected with a rubber tube and a funnel. In this manner it is possible to

introduce up to fifty quarts of water into the rumen of cattle within the course of several hours (Deffke); it may be well to add 8 to 10.0 gm. of hydrochloric acid or 8 to 15.0 gm. of creolin, lysol, resorcin. However, if there is any obstruction of the omasum or an obstruction between the sections of the stomach the introduction of such quantities of water acts unfavorably (Engesser). In obstinate cases of obstruction of the rumen or reticulum there remains as a means of last resort the incision of the rumen (see page 86); after it has been made, it is possible to clean out the rumen and then to irrigate the obstructed omasum.

In general disturbances (fever, depression) Bissauge uses, besides stimulants (caffein, strychnin), hydrogen superoxide (12% intravenously) or makes an intravenous infusion of physiological serum (2 to 4 liters) in order to detoxicate the blood.

The treatment of poisoning by turnips in cattle consists in stopping the feeding of turnips and the administration of mild laxatives, in cases of severe diarrhea also in administering large doses of creta alba and cortex quercus (Schmidt).

In atony of the stomach in young calves, Imminger achieved a rapid cure by irrigation of the rumen combined with proper diet. Heidrich treated successfully the regularly recurring tympanitis in 8 to 10 and 12 weeks old calves with antiparesin (0.1 gm. veratrum and 0.1 gm. arecolin, each dissolved in 1 liter of a decoction of linseed, and of this alternately every 2 hours a medium-sized cupful) followed by after treatment with anti-scours (Antiruhr).

Irrigation of the rumen in calves is, according to Imminger, performed as follows: A trochar 10 cm. long with a canula 8 mm. in diameter is provided with a rubber tube 1 meter long, carrying on its free end a funnel. The puncture is made with the trochar which is then withdrawn and the rubber tube is attached to the canula. A 3% warm common salt solution (30 to 35° C.) is allowed to run into the rumen until the fluid remains in the funnel. The solution is left in the rumen for some time, then the rubber is detached from the canula, but the latter is left in place in the rumen. Next the calf, whose hind and front legs have been tied together in pairs, is lifted by two or three persons upon its left side on two tables, placed side by side, so that there is left an opening of a few inches between them which will permit the canula to be in the free space. The fluid will at once begin to be voided and this may be assisted by mild pressure upon the upper side of the calf. Finally the canula is slowly withdrawn, so that all fluid which had been introduced can run out. After the removal of the dirty brown, stinking irrigation water, the rumen is filled a second time, but the fluid is now not withdrawn, but is, by proper massage, pressed into the other two sections of the fore-stomachs, the abomasum and the small intestines. After the contents of the rumen have been diminished in this manner, the canula is removed. The next day there is moderate diarrhea with fetid defecation and then recovery takes place.

Acetonemia in Cattle. Under this name Joehnk described a peculiar disease in cows which was termed by Janssen "chronic gastro-intestinal catarrh of cows following calving" and by Bass "insidious indigestion of milk cows."

Occurrence. The disease is observed in well-nourished cows as a rule after the third to sixth calf and especially (in 81% of the cows according to Eckelund and Engfeldt) in small herds. It usually makes

its appearance during the period when the animals are stabled, most frequently between the eighth and seventeenth days, but also two days to several weeks or even several months after parturition, and occasionally a few days before calving, especially during and following a drop in the barometric pressure or during a low barometric pressure (Kroge-Petersen). Repeated attacks have also been observed following subsequent parturitions (Joehnk).

Etiology. The real cause is only a matter of conjecture. Nissen, Hoeg, Veenboos and Ten Hoopen considered the disease as chronic parturient paresis partly due to the absorption of aseptic lochia or to toxins formed in the udder. Bass believes it to result from feeding too rich and unaccustomed feed which is too exacting for the mucous membrane of the abomasum, while Joehnk thinks it is due to processes of decomposition in the stomach and intestinal tract accompanied by *abnormal catabolism of protein. According to Eckelund and Engfeldt* it is the result of like disturbances in metabolism in good milk cows, consisting of excessive catabolism of fat and protein in the body and increased production of acetone compounds (beta-oxybutyric acid, diacetic acid, acetone) and in transmigration of fat to the liver to be converted into sugar, thus resulting in enlargement and fatty infiltration of the liver. The circumstance that increased production of acetone compounds in the body usually appears in conjunction with insufficient combustion of carbohydrates reminds one of the possibility of a decrease in the functional capacity of the adrenalin system as a result of too great exhaustion, and possibly increased tonus of the vagus and insufficient conversion of the glycogen of the liver to sugar following parturition and the inception of milk secretion, a condition which, according to Knoepfelmacher, is probably the basis of the clinically somewhat similar recurring vomiting in children suffering with acetonemia.

Eckelund and *Engfeldt and Joehnk construed the disturbance* as the result of acidosis due to an increased production of diacetic acid and beta-oxybutyric acid with neutralization of the alkalies of the blood, *thus bringing about an accumulation of stupefying carbonic acid* and other waste products of metabolism in the tissues. In this connection, however, it is also deemed possible that it is due to the action of acetone, which in small quantities is an excitant, in large quantities a depressant.

Symptoms. The animals at first show depressed appetite, become rather selective in their food consumption, refuse especially grain and good hay, at first only partly, finally totally, while straw bedding, heath and bad, moldy feed is readily consumed. Sometimes symptoms of pica appear. Accompanying the decreased consumption of hay is a decrease in the circumference of the abdomen (falling in at the flank, *tucked-up abdomen), rapid emaciation, slow rumination, during* which some animals suddenly stop mastication only to resume it after a short pause. Light constipation and straining during defecation are also observed. The animals are also *easily excited, continually looking* around and listening as soon as someone enters the stall. Frequently, however, the animals are in a condition of semiconsciousness, show depression of the sensorium and have a swaying gait. As a rule the frequency of respiration is reduced (up to 8 per minute) and there is pronounced abdominal respiration. Not infrequently the rate of the pulse is also reduced (44 to 50 per minute). In severe cases the

heart beat becomes pounding and the pulsations of the abdominal aorta can easily be detected. A further frequent phenomenon is an increase in the size of the liver as determined by percussion, accompanied by increased resistance and sensitiveness to pressure in the region of the liver. Particularly noticeable is the unpleasant, sharp, aromatic, sweetish, chloroform-like acetone odor of the breath which can also be detected by opening the mouth of the animal, in the urine, in the gradually decreasing amount of milk and in the air of the barn. Simultaneously an increase in the amount of acetone can be detected in the urine and milk. The urine gradually becomes amphoteric and finally acid in reaction, appears watery, and its specific gravity falls to 1005 and below.

Course. The disease may last several weeks and without affective treatment terminate in cachexia and death.

Diagnosis. This is based upon the appearance of light digestive disturbances together with a peculiar change in the behavior of the animal, as well as the acetone odor of the breath and all excretions and secretions of the body shortly after parturition in good milk cows, but especially upon the detection of an increased amount of acetone in a reliable sample of urine and milk. Particularly will there be observed a turbidity of a precipitation of iodoform upon the addition to the urine (or, better, to its distillate) of Légal's reagent (iodin 1.2, potassium iodide 1.8, distilled water 30) and thereupon an excess of 10% potassium or sodium hydroxide (Lieben's test); furthermore, a violet ring after mixing 15 cc. of the distillate of the urine with 20 drops of a mixture of equal parts of glacial acetic acid and a 10% solution of sodium nitroprusside and superimposing the mixture with ammonium hydroxide. (In the urine of healthy cattle 0.2 to 4.4 mg. acetone per liter has been found, in milk 1.45 to 2.5 mg.) Puerperal hemoglobinuria occurring under like circumstances differs from acetonemia by an elevation of the body temperature (40 to 41° C., 104 to 105° F.) at the beginning, followed by a drop to below normal; by increased, strongly pounding heart beats, pronounced paleness of the mucous membrane, and especially by the appearance of hemoglobinuria simultaneously with a reddish-brown discoloration of the milk. In light cases of parturient paresis one finds, according to Eckelund and Engfeldt, besides indigestion only a moderate increase of acetone and beta-oxybutyric acid in the urine, while the acetone content of the milk remains normal. Compared with the usual forms of atony of the forestomachs, the emaciation in acetonemia has no relation to the slight disturbances in digestion. There is also noticed a certain excitement, often a sleepy behavior, and acetone odor of the breath as well as of the excretions and secretions.

Treatment. Exercise and pasturage, as well as the administration of sodium bicarbonate (daily up to 50 gm. per 100 kg. body weight) and chalk (same amount), have a favorable influence. Joehnk recommends the administration of the following mixture, repeated every 8 hours: Extract of aloes 25 to 30 gm., sodium bicarbonate 350 to 400 gm., sodium sulphate 75 gm., radix gentiana 75 gm. Furthermore an attempt should be made in severe cases to check the formation of acetone through increased combustion of carbohydrates in metabolism by feeding easily digested carbohydrates such as boiled mashed potatoes, syrup, molasses, etc. In all cases, especially those resembling

parturient paresis, the udder should be inflated with air, whereupon the condition may at first grow worse (twitching of the muscles and eyelids, convulsions, champing of the jaws, salivation) but is soon followed by recovery.

(e) Foreign Bodies in the Fore-stomachs. Corpora aliena reticuli, ruminis et omasi.

(*Traumatische Hauben-, Haubenzwerchfellentzündung, Traumatische Indigestion, Traumatische Pansenlähmung* [German] [EBER].)

This is a collective term comprising disturbances in digestion partly due to gastroperitonitis resulting from pointed foreign bodies and partly to inhibition of movements or obstruction of the fore-stomachs due to blunt foreign bodies.

Occurrence. Foreign bodies and diseases caused by them are met with especially frequently in cattle; in the other ruminants (goats, sheep, bison) only seldom, yet proportionately *most frequent in goats.* Particularly cattle having female attendants, and cattle of poor owners are affected, as these animals have usually the best chance of taking up foreign bodies. Still more frequent are cases in the barns of tradesmen and factories where work is done with nails and needles (shoemakers, needle makers, iron workers, spinning establishments) as well as in industrial regions, in the vicinity of large cities (use of the sweepings for fertilizer) and in years of scant harvest when all kinds of substitute feeds are used. Sometimes gastric disturbances due to foreign bodies appear in a herd in an almost enzootic manner.

According to Eber's statistics of 235 correctly reported cases of paresis of the rumen (overfilling of the rumen, primary *and secondary gastric atony) in cattle, 17.9% were due to* foreign bodies. Of 50 cases of traumatic gastroperitonitis compiled by Udall only 7 occurred in summer. It occurred in animals ranging in age from 2 to 8 years, and was caused in 18 cases by pieces of wire, in 19 cases by nails, in 2 cases by needles, while in 10 cases by foreign bodies which could not be found. In 110 cases reported by Wenger there were found in 48 cases pieces of wire, in 46 cases nails, in 10 cases sewing needles or pins and in 6 cases hairpins.

Etiology. The frequent occurrence of foreign bodies in the fore-stomachs of cattle depends upon the habits of these animals to lick all possible objects accessible to them and often to swallow them. Besides various objects are found in the food containers, particularly in the poorly kept barns of small farmers, which have gotten there with the garbage, the sweepings, etc., or which are derived from the wardrobe of female *attendants. Such objects are then easily swallowed with the* feed, the more so since cattle chew their food only very superficially and coarsely before swallowing. (There is a case on

record of a perforated but otherwise complete egg shell being *found in the rumen of a cow.*) Besides, *the long papillæ of the* tongue projecting posteriorly prevent foreign bodies from falling out of the mouth. But even with good care the feed may contain foreign bodies such as pieces of wire in pressed hay, nails or pieces of wire which may have been derived from burned wood and may have been swallowed in the pasture.

Smaller foreign bodies vary a good deal as to their nature and form; most dangerous are those of iron because they are not changed by the juices of the fore-stomachs. Needles, pieces of wire, nails, blades of knives, pieces of scissors are found most commonly; less common and less dangerous are splinters of wood, larger pieces of wood, leather, cloth, balls, pieces of money and roots.

Licking of the wall of the barn, pasturing on marshy places with the ingestion of marshy or dirty hay often brings sand in large quantities into the fore-stomachs and sometimes causes extensive disease of cattle, particularly in inundated territories (observed by Marconi in buffalo calves and by Müller and Prietsch in cattle). Drinking in shallow bodies of water may also cause overfilling of the fore-stomachs with sand.

Clinically, concrements also act as foreign bodies, of which particularly the hairballs (bezoars of animal hair, zootrichobezoars, pilobezoars, pilo concrements, aegagropili) are not infrequently formed in the rumen and in the reticulum of cattle and also in other ruminants while licking their own bodies or that of a neighboring animal. The swallowed hair or wool fibers are formed into spherical or egg-shaped bodies by the rolling or churning motion of the fluid or semi-fluid contents of the rumen during the sucking period, while on soft feed or during the regular consumption of large quantities of fluid, similarly as the seaballs are formed by the rolling and churning motion of the water in shallow water (Francke). They sometimes reach tbe size of the fist, with an outside shell consisting of mucus and inorganic salts, or have the appearance of skin with a dense growth of hair. They are formed most frequently in adult cattle, sometimes in calves and lambs, particularly after eating wool or following itching skin diseases.

Fetal hair-balls are also sometimes observed in new-born animals, especially in calves, which may increase in size after birth by the addition of swallowed hair. According to Francke they originate in the amniotic fluid from the cast-off hair of the fetus being formed into small balls by the shifting of the fluids through movements of the fetus and being later swallowed by the fetus. In this manner the presence of hair-balls the size of chestnuts in calves born dead (Schell) and the size of an apple to the size of a fist in calves 10 to 21 days old (Bonnet, Hoffmann, Mégnin) can be explained.

Feed balls originate in a similar manner to hair-balls and

are made up either of hairs of plants or plant fibers (phytothricobezoars, phytobezoars, and phyto concrements) containing inorganic salts and other particles of food, for they are only conglobates of undigested parts of plants (Joest). They are formed during continued feeding on cottonseed cake made from undecorticated cottonseed or by feeding woody turnips, by the matting together of the undigested remnants of such feeds (Moussu). They are seldom found in the fore-stomachs of lambs but usually in the abomasum.

Pathogenesis. Blunt foreign bodies only rarely lead to bad consequences for the animals; but these occur when the foreign bodies are pressed into the communications between the sections of the stomach and intestines or into the pylorus; or when they are as large as a nut, so that they prevent free communication between the fore-stomachs or between abomasum and intestines, or if they prevent the motions of the fore-stomachs. In all these cases the removal of the feed-mash and of the gases suffers more or less and the muscles of the fore-stomachs become relaxed (see page 96). Pressure exerted upon the wall of the stomachs by heavy foreign bodies may bring about nutritive disturbances on the parts of the affected wall, and finally inflammation which may spread to the peritoneum.

The effect of **sharp** or **pointed bodies** depends upon their nature, form and length and also upon their location in the stomachs. Larger foreign bodies are, in general, less dangerous than smaller ones, because they do not so easily get from the rumen into the reticulum and may remain for a long time embedded in the feed-mash in the rumen without producing any evil effect. (Bergmann found a kitchen knife in the rumen which had been there three months without causing any damage.)

Foreign bodies, as a rule, become wedged into the wall of the comparatively small but vigorously contracting reticulum, while the rumen, the omasum and the abomasum are rarely injured. Pointed bodies of inconsiderable length (nails, pieces of wire, hairpins), particularly if provided with a head or bent over, get more or less deeply into the wall of the stomach, but they do not penetrate further. However, long and pointed bodies, smooth along their whole extent (such as long nails or pieces of wire, darning, embroidery, suturing or tobacco needles) will be transported not only to neighboring, but even to distant organs by the contractions of the stomachs and the diaphragm and by the abdominal pressure (especially during parturition and during restraint before an operation) or by pressure of the uterus if this is well along in gestation. Since perforation usually occurs in that part of the wall of the omasnm which is directed towards the diaphragm, these foreign bodies usually penetrate in the direction of the diaphragm or heart.

Perforation and continuous irritation of the wall of the stomach first causes pain, which, however, is fairly acute only if the penetration took place more or less rapidly. The injury of the wall of the stomach opens the way to the bacteria which are contained on the foreign body and in the gastric contents, and in this manner causes a circumscribed inflammation of the wall of the stomach and the peritoneum around the puncture. Depending upon the rapidity of penetration of the foreign body, the inflammatory process develops either rapidly, with profuse formation of fibrin upon the serous membrane, or slowly and with slight fibrin formation, forming adhesions between the diseased wall of the stomach and the adjacent parts. The thick layer of fibrin will now, in spite of a later complete penetration of the wall of the stomach by the foreign body, prevent the escape of microorganisms into the abdominal cavity. In case the foreign body has quickly penetrated the wall of the stomach before the formation of a layer of fibrin has taken place, or in case the microorganisms have penetrated into and continued to grow in the fibrin, a general acute peritonitis may develop. A similar protective action upon the peritoneum distant from the seat of injury is afforded by the connective tissue and adhesions taking the place of the fibrin when the irritating process is of long duration. A perforating foreign body may exceptionally injure a larger blood vessel.

The motions of the fore-stomachs are interfered with both by the injury to the gastric wall itself, and also by the acute inflammatory process, and later on mechanically by adhesions which are formed (Eber's traumatic paresis of the rumen).

Cases in which the inflammation does not develop very slowly, afford the opportunity for the absorption of bacteria and bacterial endotoxins into the circulation; in this manner fever is induced and also leucocytosis through stimulation of the bloodmaking organs. In old cases such an absorption may be prevented entirely by the fibrous connective tissue around the foreign body, which is poor in blood vessels.

Anatomical Changes. Blunt foreign bodies are, as a rule, *found either imbedded in the feed-mash in the rumen or they* are wedged into the openings of the stomachs or the pylorus. Sand or gravel may become deposited on the internal surface of the rumen, more rarely upon the other sections of the fore-stomachs. (Wilhelm found 120 pounds of sand in the fore-stomachs, the walls of which had become perfectly hard, socalled "*Magenversandung*" [German] Saburra.) Occasionally one sees inflammatory changes of the mucosa of the fore-stomachs, exceptionally a circumscribed peritonitis (Roeder, Wilhelm).

Pointed foreign bodies cause changes preferably in the wall of the reticulum (traumatic inflammation of the reticulum,

reticulitis traumatica). On post-mortem examination loss of substance, with hyperemia in the neighborhood, small hemorrhages, also purulent inflammation are found, sometimes in the mucosa only, at other times in the deeper tissues of the gastric wall, or there may be a cicatrix only or a fistulous tract surrounded by cicatricial tissue. In other cases the *foreign body has penetrated further into the wall of the stomach* or has perforated it, then a circumscribed fibrinous peritonitis is found in the immediate neighborhood of the injury, and an agglutination or adhesion of the reticulum to the diaphragm (traumatic diaphragm-reticulum inflammation) or to the rumen. Frequently one will find a fistulous tract in the cicatricial adhesions which communicates with the reticulum, or the cicatricial tissue includes tough-walled abscesses containing usually ill-smelling pus which is occasionally mixed with food particles. The fistulous tract usually contains the rusty or dark discolored foreign body, but this may also have become disintegrated.

The pointed, long and smooth foreign bodies frequently penetrate from their first place of perforation into other organs, most frequently towards the heart, more rarely towards the *lungs, liver, spleen, muscles of the thigh; there are then formed* cicatricial bands leading to the reticulum, which may be interrupted by abscesses. In those very rare cases where a foreign body has made its way to the outside world, there is formed a thick-walled fistulous tract leading from the region of the ensiform cartilage to the reticulum.

Foreign bodies are rarely found in the omasum or abomasum; they get there from the reticulum and sometimes leave the body through the intestines. Exceptionally a foreign body may travel over another route and penetrate through the left hypochondriac region, through the flank, or through the muscles of the hind leg; the road traveled is then also indicated by cicatricial tissue or by a fistulous tract.

Small hemorrhages at the site of the injury are common and the grayish discoloration in the newly formed connective tissue is due to these extravasations. Exceptionally internal hemorrhage may be noticed (Eggeling, Bräuer, Harms, Holterbach), then the reticulum and the next sections of the stomach, also the intestines contain blood coagula. Sometimes we encounter purulent ichorous peritonitis or pleuritis in consequence of the breaking of an abscess in the neighborhood of the reticulum. General purulent or sero-fibrinous peritonitis may also be developed if the gastric wall has been perforated without the prior formation of sufficient preliminary agglutination or adhesion; in such cases bacteria enter the peritoneal cavity from the reticulum. Sometimes we find on post-mortem metastatic foci in the internal organs.

Symptoms. Blunt foreign bodies sometimes produce an atony of the fore-stomachs (chronic dyspepsia) which cannot

be distinguished from other forms of gastric atony and which therefore can only be diagnosticated if the history of the case furnishes some data. After the ingestion of sand (socalled "*Magenversandung*") or gravel, there are also present symptoms of chronic gastric atony (see page 109), and in some cases the wall of the rumen feels as hard as a rock; the sick animals constantly make empty masticatory movements, saliva dribbles from the mouth, the back is curved and a painful groaning is frequently heard (Krichels). The feces sometimes contain sand. In cases of sand in the rumen Wucher saw a paresis similar to parturient paresis of cows. In severe cases the disease leads to exhaustion and sometimes ends fatally after two to three weeks, but it often lasts for months.

The sudden stoppage of the openings between the forestomachs or the pylorus by foreign bodies, especially hair or food balls, occasionally occurs in calves and lambs and exceptionally in adult cattle. Such stoppage causes bloating which appears suddenly and increases rapidly, great restlessness, spreading of the front legs, loud groaning, anxious expression, later on listlessness, almost complete inability to walk, increase of pulse and respiration, even eclamptiform attacks, sometimes vomitory movements which may cause the expulsion of the obstructing hair or food ball, and may in this way bring about a rapid recovery. Nevertheless, young animals usually succumb in one to two days (Richards saw three to five lambs die daily in a herd of 120).

The penetration of **pointed bodies** leads to inflammation of the stomachs (traumatic gastritis and inflammation of the diaphragm) and causes profound gastric disturbances, and in those cases which have not developed slowly, symptoms pointing to tenderness of the stomachs and of the diaphragm.

The time interval between the ingestion of the foreign body and the appearance of the gastric disturbances is very variable according to the nature of the foreign body. Two oxen experimentally received some medium sized nails pointed at both ends; they were introduced through the esophageal sound. One animal showed symptoms after twenty-four hours, the other only after four days (Marek). Koppitz inclines to the belief that at least four days elapse on an average between the ingestion of a foreign body and the appearance of the gastric disturbances, because the foreign body, which is usually introduced into the rumen, will pass into the reticulum only after this time. If the foreign body is accidentally taken up with mashy or fluid feed, it may get directly into the reticulum and penetrate into its wall.

In cases of rapid development in one-half to two-thirds of the cases the symptoms appear suddenly with severe digestive disturbances and without apparent cause, particularly without faulty feeding and under normal conditions of care. The animal will suddenly refuse feed, will later begin to eat again, but for a longer time the consumption of food is very inconstant. Rumination either ceases entirely or is very irregular and slug-

gish. Under some circumstances it may be difficult for the animal to regurgitate, the morsels having more than appearance of strangulation (Wegner). In about one-third of the cases chronic meteorism is observed, which condition may vary within a few hours and in the degree of intensity, but as a rule is most intense soon after feeding. A frequent phenomenon is also a moderate degree of distention of the rumen with feed even between meals so that the animal constantly appears as though well fed. Only seldom does the rumen appear excessively filled with feed (taking sick immediately after feeding), while a gaunt appearance is more frequently observed. In the majority of cases the movements of the rumen are greatly depressed or entirely absent, so that slight movements can be detected only intermittently. Sounds of the rumen behave in a similar manner. As a rule the animal is constipated, while later small blood clots or blood streaks may be mixed with the feces.

In contrast to the simple atony of the stomach with similar symptoms of disturbance in digestion, in this condition symptoms of pain are observed from the beginning. The animal displays much anxiety in its whole behavior, moves reluctantly, stands with arched back, placing the feet well under the body, issues plaintive sounds (frequently audible outside the barn) when lying down, while getting up, during defecation and the passing of urine, when making short turns, with every step it takes, particularly when going down hill, and its gait is careful and stiff. The manifestation of pain is increased when the animal is forced to stand with the hind legs higher (Imminger). Intermittent colicky symptoms appear (striking at the abdomen more frequently with the left foot [Wegner], stepping about restlessly, switching the tail, etc.). Often a decided trembling of the involuntary muscles is observed (Oppermann), sometimes of the whole body especially when standing for a long time. A very important symptom is sensitiveness to pressure in the region of the xiphoid cartilage. When strong pressure is exerted on this point with the fist assisted by the elbow resting on the knee of the flexed leg, or if this region is struck a short, strong blow, the animal will try to hook or kick the operator and will always groan (in Wegenheuser's case the cow flinched). Oppermann found the method of bringing pressure to bear alternately upon the region of the xiphoid cartilage and the back very advantageous to force the animal to utter sounds of pain. A similar behavior of the animal can sometimes be observed by percussion or palpation at the line of the diaphragm along the costal arches near the xiphoid cartilage.

As a rule general depression is observed, also a slight or medium elevation of the body temperature (39.7 to 40.8° C., 101.5 to 105.5° F.), which, however, frequently appears only as a trembling of the muscles, hair standing erect, hot or cold ears, horns, muzzle and legs, without a simultaneous rise in body

temperature. Respiration is accelerated and shallow, seldom labored. Intermittently a short, dry, painful cough is observed. Independently of the behavior of the body temperature and in contrast to indigestion due to other causes, the pulse is always unusually accelerated (up to 72 to 100 per minute), while after exercise of short duration the heart beats so rapidly that the pulse can no longer be counted. Combined with this, as a result of great weakness of the heart, paleness and cyanoses of the mucous membrane and slight edema of the eyelids can be observed, as well as sometimes a medium jugular pulse in the region of the throat and dewlap. Brenner observed pronounced variations in the blood pressure of the arteria coccygea. Neidinger observed albuminuria in 29 out of 33 cases, Schultz as well as Bider neutrophilic leucocytosis in the blood. The milk flow is gradually or suddenly pronouncedly decreased and may cease entirely. The animal rapidly loses flesh, grows weak and appears tired. Often the animals lose as much as one-tenth or more of their body weight within a few days. Musterle observed epileptiform convulsions in one case.

The prolonged and severe disturbances of digestion and the abscesses which often form in the meantime cause an ever-increasing weakness and emaciation, and in milk cows the milk flow ceases. In the further course of the disease there is a gradual decrease and final disappearance of the pain, absence of fever, permanently or for varying periods of time, and a lowering of the rate of the pulse and respiration. The symptoms finally simulate those in cases of medium severity which begin without pain.

Deviation from the usual course may sometimes be observed, as occasionally the affection may begin suddenly with gagging and vomiting, in which case blood or feed mash may be ejected from the mouth and nose (Mueller, Wilhelm, Sequens, Holterbach) and the animal may suffocate through aspiration of the feed mash (Sequens). Likewise, in exceptional cases, the passing of liquid or coagulated blood from the rectum may be observed (in a case of Harms the coagulum plainly showed the impression of the mucous membrane of the reticulum), in which case the hemorrhage is only temporary or soon leads to the death of the animal, as in the case of the vomiting of blood that is sometimes observed (Eggeling, Holterbach, Rothaar). In a case described by Eber one animal showed paralysis of the tongue and inability to swallow and had a puncture of the esophagus at the point of implantation of the wound. More frequently the disease begins with symptoms of overloading of the rumen or acute meteorism when the injury occurs at a time when the rumen is full. The traumatic gastroperitonitis of breeding animals shows, according to Wenger, almost regularly a deviation from the usual clinical course, since the foreign body, still free in the reticulum and following the law of gravitation, will, during service, under the influence of the thereby increased intra-abdominal pressure, become lodged in the posterior wall of the reticulum, whereupon th inflammation will begin at the posterior or lateral wall of the reticulum. In such cases there will be slight fever, slightly increased activity of the heart, difficult breathing, depressed appetite, always some rumination, slight bulging of the flanks, increased tension of the abdominal muscles, weakness, emaciation and edema under the abdomen in front of the scrotum.

In the **slow development** of the inflammatory processes during the course of the slowly progressing penetration of a somewhat blunt foreign body which may not entirely penetrate

the wall of the reticulum, pain is entirely absent or insignificant. A slight indigestion which may simulate a harmless trouble is *noticeable. It reaches its climax in 2 to 3 days and then disappears* permanently or temporarily, but as a rule permanently. More frequently disturbances of digestion resembling atony of the fore-stomachs will be observed. They are, on the one hand, characterized by spontaneous improvement and aggravation, that is, by relapses through weeks and months without apparent cause or following parturition, transportation, service, etc., or, on the other hand, by resistance to the treatment usually applied in other diseases of the fore-stomachs. According to the *author's own observations, one not infrequently finds on* percussion a circumscribed dull area of varying size in the *region of the reticulum (Fig. 8),* more frequently on the left side or on both sides anteriorly and posteriorly to the edges

Fig. 8. Dullness *(d)* over the region of the reticulum in traumatic inflammation of the reticulum and the diaphragm.

of the lungs, from which on puncture an ichorous fluid can be obtained (from abscesses proximate to the wall of the chest or abdomen).

General symptoms and changes in the action of the heart and respiration may be absent or not pronounced. In spite of this there is a decided decrease in milk flow and frequently albuminuria and leucocytosis, while emaciation and weakness are observed only in some of the cases.

Complications arise frequently in consequence of the wandering of the foreign body, exceptionally also in animals which at first did not show any symptoms at all. Comparatively frequent is traumatic pericarditis, occasionally traumatic splenitis (according to Wegener in about 14% of cases) or a traumatic hepatitis; in both cases the disease is apt to take a

peracute course, causing high, continuous fever (40.5-41.5°C.) with very rapid pulse and marked debility. Disease of liver or spleen is recognized only by enlargement of these organs and sensitiveness to pressure. The cases of traumatic splenitis and hepatitis, occasionally those of simple traumatic gastritis, may be complicated by general acute or chronic peritonitis (dullness on percussion, audible splashing, visible undulation when short, strong pressure is exerted upon the abdominal wall, positive exploratory puncture) in acute cases showing a sensitiveness on pressure upon the abdominal wall, in some cases ichorous inflammation and furthermore, under some conditions, collection of putrefactive gases between the rumen and the superior abdominal wall (so-called false tympanitis). More seldom a traumatic pneumonia or pleurisy develops, exceptionally with fatal hemorrhages (Wyssmann) or with evacuation of ichorous pus through the nose, following the rupture of an abscess located between the reticulum and the lung or within the lung itself (Zieger). Sometimes the scene is closed by a general infection with septicemia or pyemia, sometimes again the bacteria are carried along and deposited in distant organs, for instance, the joints where they then produce local inflammatory processes.

Finally a more or less painful swelling of doughy or firm consistency develops in rare cases in the region of the ensiform cartilage, behind the left elbow or in the left region of the lower ribs, exceptionally also on more distantly situated places of the body, which breaks open after a time and discharges pus or rather an ill-smelling ichorous fluid mixed with feed material through the opening, and sometimes the foreign body is also discharged through this opening. The fistula so formed later on sometimes discharges particles of masticated food from the stomach and closes only gradually. It may close after weeks and months and lead to final complete recovery; or a cachectic condition may develop gradually, in consequence of gastric disturbances and extensive adhesions, which finally leads to death. In a case of Zieger the pointed end of a thick iron spike emerged behind the right elbow without having caused previous symptoms of disease or the formation of abscesses excepting a transient disturbance of appetite some five months before. In Wenger's case a broken whip-handle produced abscess formation along the edge of the sternum, similar to a case observed by Leve, which was caused by a piece of wire. In Mohr's case a slat, 55 cm. long, from a feed rack, after assuming a vertical position between the xiphoid cartilage and the ninth to tenth rib caused an inflammatory swelling near the latter point on a level with the hip joint.

Course. Obstruction of the gastric openings caused by dull bodies is of short duration and either ends in recovery within

a few days, that is, if the foreign body is removed either by vomiting or by returning into the interior of the stomach, or death rapidly follows if the obstruction persists. Chronic gastric atony due to dull foreign bodies usually lasts for weeks and months; in the meantime the animals become more or less emaciated or they die within nine to seventeen days if the stomachs are filled with sand or similar material (Krichels).

The inflammation which is caused by pointed or sharp foreign bodies subsides within a few days or weeks if the gastric wall has been only injured but not perforated, or there may be established chronic gastric disturbances which are due to adhesions. If, however, the pointed foreign body has perforated the stomach wall, complete recovery may occur in rare instances if this foreign body slips back into the fore-stomachs or if it pushes itself into the outside world; nevertheless, as a rule the animal exhibits gastric disturbances until death occurs. In certain cases a shorter or longer period of improvement is followed by an aggravation because the foreign body which has slipped back into the stomach again penetrated into the gastric wall. Such variations in the picture of the disease may occur repeatedly. The possibility of recovery seems to be excluded when symptoms appear pointing to inflammation of the neighboring organs. A fatal issue may take place within a few days if the foreign body penetrates rapidly, but the disease usually lasts for weeks and months.

The appearance of complications has always a bad prognostic meaning. Fever likewise is an important factor and a long continued elevation of temperature points to progressive deeply penetrating inflammatory processes.

Diagnosis. The exact origin of gastric disturbances due to dull foreign bodies can only be surmised from the history of the case, although sudden bloating excites suspicion as to hair and food balls in the stomach, particularly if the animals have recently shown strong inclination to licking. Exceptionally the correct diagnosis can be made if the foreign bodies are found in vomited material or in the feces. Lodgment of hair balls in the cardia of calves may be assumed, according to Schauber, if bloating does not yield after the administration of tincture of veratrum (30 to 40 gm. three times daily).

The clinical picture of gastritis and peritonitis due to pointed and sharp foreign bodies, is characterized by the following symptoms: The sudden appearance of severe, obstinate and changing digestive disturbances without demonstrable cause; frequently more or less bulging of the left flank, retarded or feeble motions of the rumen, spontaneous expressions of pain, especially in the recumbent position, on making short turns, on going down-hill, during activity of the abdominal wall, after the posterior part of the body has been raised, which will aggravate the condition, sensitiveness to pressure in the region of the

reticulum, unusual acceleration of the heart action after brief motion; rapid emaciation. None of these symptoms can be accepted as pathognomic, if isolated, except the accidental appearance of the foreign body beneath the skin. Careful systematic and repeated examination and observation of the complicated clinical picture usually make possible a positive or at least a probable diagnosis. In other cases the advent of complications (pericarditis, pleuropneumonia, peritonitis, splenitis, hepatitis) in the later course, often indicates the correct diagnosis.

In consideration of the difficulties in establishing the diagnosis, certain auxiliary means should be applied in suitable cases. First of all an exploratory abdominal puncture behind the ensiform cartilage, between umbilicus and milk vein (which operation is best performed with Salomon's trocar), usually

Fig. 9. Modified Salomon's trocar.

yields, according to Sallinger, a serous exudate during the initial stage. According to the experience of the authors, it can afford valuable information in more advanced cases with circumscribed areas of dullness, by revealing the presence of ichorous exudate. The presence of neutrophile leucocytes in the blood, in general suggests suppuration, but in the presence of obstinate digestive disturbances it indicates traumatic gastritis, if no other cause has been found for the leucocytosis. In cases which develop very slowly, and in encapsulated abscess, *leucocytosis may be entirely absent.*

In cases of fever above 40° C. (104° F.) the persistence of an accelerated pulse in spite of a fall in body temperature after the administration of 50 to 100 gms. antifibrin per day is *indicative of traumatic gastritis*, according to Oppermann. Furthermore, the aggravation of the condition as well as the manifestation of pain when elevating the hind legs will, in cases of traumatic gastritis of recent origin, make the diagnosis more easy.

In agreement with Johne's suggestion, many authors (Eber, Holterbach, and others) recommend remedies which stimulate contractions of the stomach, like Tartar. emet. (8-10 gm.), Eseridine tartrat. (0.2 gm. subcut.), Veratrin. sulph. (0.10-0.15 gm. subcut.) Arecoline hydrobrom. and Veratrine sulph. (alternately 0.1 gm. of each in tablets, by mouth); also Arecovetrol and Antiparesin (see page 85).

According to Joest and other Swiss veterinarians, cattle with traumatic gastritis will react after the administration of

hydrochloric acid (1 to 2 tablespoons), similarly as they will in cases of ulcer of the abomasum (aggravation of the condition [Salvisberg]). The following symptoms are considered as pointing conclusively to the presence of traumatic gastritis: spontaneous manifestations of pain (moaning, stepping about restlessly), increase of digestive disturbances, aggravation of the general condition. The want of appetite, absence of rumination and of the motions of the rumen are also believed to be diagnostic without other signs being present, when they occur in response to these remedies. But this method is not absolutely reliable. The disappearance of manifestations of pain with transitory improvement after the administration of the remedies mentioned may be noted in old cases of traumatic gastritis in consequence of increased contractions of the stomach, if no extensive adhesions exist and no abscess is present. On the other hand, these remedies may cause pain in healthy cattle and especially in tuberculous cattle and may diminish the appetite for a few hours. They are therefore useful in the diagnosis of cases which have not yet become chronic, in which the more vigorous contractions of the stomach may irritate the nerve endings which have become sensitive in consequence of the acute or subacute circumscribed peritonitis, and they may also cause the foreign body to move forward. These measures should be reserved for special cases, as they may easily lead to a lasting aggravation of the traumatic gastritis and thereby prevent a cure which might be possible otherwise. Bass condemns the employment of the remedies mentioned as unjustified with regard to the cases of recent origin with only slight adhesions in which such a procedure could easily frustrate the desired cure by aggravating the condition. In more advanced cases with organized adhesions and encapsulated abscesses this procedure is, as a rule, not considered dangerous. Whether it is preferable to use the chemical test for iron foreign bodies in the contents pumped from the stomach, as recommended by Kreutzer as the result of his experiments, or the test by means of a digestion cell introduced into the stomach to establish an early diagnosis of traumatic gastritis, must be decided by the experience of the practitioner.

In differential diagnosis certain cases of overfilling of the rumen, acute bloating and atony of the fore-stomachs due to other causes are to be considered. The important points in differential diagnosis have already been mentioned (see pages 84, 90, 95). General acute peritonitis and gastritis take an acute course with high fever, and grave general disturbances; there are, however, cases in which even these affections cannot be absolutely excluded. Tuberculosis of the lungs can be excluded if certain physical signs which may possibly point to it, are only found in the posterior and lower portions of

the thorax, and perhaps only on one side, and if gastric disturbances have preceded the preliminary affection. Sometimes the tuberculin test may decide the question. Tuberculosis of the mediastinal glands can easily be distinguished from traumatic gastritis.—A complicating pericarditis, pneumonia or pleurisy might be confounded with tuberculosis. However, secondary complications of traumatic gastritis are preceded by gastric disturbances, and later they remain in the foreground of the clinical picture. However, all circumstances must be considered in each case and the possibility of tuberculosis must be kept in mind.

Prognosis. It is always necessary to be cautious in making a prognosis, because the termination of the disease can never be predicted. In general the prognosis is unfavorable in the majority of cases, aside from the rather frequent instances with slight symptoms which cannot be differentiated positively from other, less dangerous affections of the digestive organs. When complications have made their appearance, recovery is quite improbable.

Treatment. The removal of blunt foreign bodies may be brought about by the same treatment that is recommended for overfilling of the rumen (page 85). In case the rumen is filled with sand, laxatives may act well, especially castor oil (500-1000 gm.) with large quantities of a mucilaginous decoction to which rhizoma of veratrum (10-20 gm.) may be added; the latter may be given alone, later on (Poetting). A heifer ill with indigestion after having swallowed a piece of rope to which a piece of wood was attached, recovered, according to Meyer, after the administration of a laxative together with Rhizoma veratri. In sandy rumen, treatment is, however, often unsuccessful. Occlusion of the gastric openings is sometimes relieved by remedies which stimulate the motions of the rumen; but in most cases incision into the rumen is required.

In digestive disturbances produced by pointed foreign bodies, treatment appears promising in cases which are not far advanced and are free from complications, as is suggested by recent reports of recoveries (Arnold, 90%; Blendinger, 97%; Nordstroem, 80%), although these cases probably include some that were not of traumatic origin, because of diagnostic difficulties. Wenger advocates early killing in cases in which the diagnosis is established. The treatment consists in raising the fore part of the body for a period of three, six or eight weeks, letting the animals fast for one or two days, and then administering easily digested lukewarm drinks which contain gruel; these are to be given three or four times daily, a large part of which, on the one hand, goes directly into the omasum, while on the other hand they do not possess any vegetable ferments having a tendency to excite movements of the fore-stomachs and therefore

are suited to give these organs a desired rest. Especially suited for this purpose are lukewarm gruels containing much mucilagenous material, etc. Arnold recommends boiled or scalded crushed oats with one-third linseed meal added; Blendinger a soup made from bread, boiled grain, linseed meal, etc.; Opperman linseed gruel with milk or gruels made from flour, bran and chopped grains. Coarse food should be withheld for one or two weeks and then be given in gradually increasing amounts. Only in case of necessity a handful of hay or of other coarse food is given in addition to the liquid food. The eating of bedding or of hay from the mangers of neighboring animals is prevented by a suitable muzzle which does not interfere with rumination, or by isolating the patient. Undue pain is relieved by 30-40 gm. of tincture of opium (three times daily with water or gruel), which drug also has a decided tendency to inhibit the movements of the stomach. Constipation or tenesmus are treated with a few doses of magnesium sulphate (Blendinger) or enemas (Oppermann). Purgatives are generally considered as useless and even harmful. Oppermann also applies wet packs to the chest. Majewski recommends blisters in the region of the xiphoid cartilage, for which purpose, according to Oppermann, spirits of mustard are less suitable than cantharides because the former produces undesirable symptoms in the respiratory tract of tuberculous animals.

The elevation of the foreparts of the body may, according to Sallinger, be accomplished by constructing a bridge 30 cm. high, supplied with cross boards and slanting backwards, or a plank or door resting on a thick piece of lumber (Arnold). Nordstroem employs a footstool, Blendinger constructs a solid, dry and even inclined plane, by means of used sheep bedding. In this case a muzzle is needless because cattle do not eat the straw of sheep bedding.

An operation for the removal of the foreign body may be considered in special cases. An incision into the rumen has been performed successfully in the presence of dull foreign bodies (Scherg, Spörer). The incision of the rumen should always be tried in the milder cases and in those not particularly far advanced since the animal may still be slaughtered even if the operation is not a success. However, in long-existing and very grave cases this operation is no longer to be recommended since it will, under these circumstances, not lead to recovery. In some cases the operative treatment of abscesses in the neighborhood of the fore-stomachs has to be considered (Moussu).

The **removal of foreign bodies through an incision** into the rumen was first proposed by Obich and it was practiced later on by Meyer for the cure of traumatic pericarditis. The operation is performed by making an incision into the left flank of the standing animal (see page 86). The hand is then introduced through the operation wound and guided towards the opening between rumen and reticulum, situated to the right and below. If the foreign body is found it is extracted. Obich was successful in four out of thirteen cases and Meyer caused in one case the disappearance of the pericardial symptoms which were already developing.

Schöberl's massage method of removing a foreign body which

had penetrated into the gastric wall, consists in placing the animal on its back. The operator then stands next to the animal on a chair and with one foot placed upon the ensiform cartilage he pushes upon it; six to ten times is usually sufficient. It is claimed that in only two out of sixty animals so treated did the foreign body fail to slip into the reticulum. Considering the fact that a sudden increase of intra-abdominal pressure may favor the penetration of the foreign body into one of the neighboring organs, the procedure of Schöberl appears to be not without danger in many cases, in spite of the fact that Schöberl and also Estor claim to have had such good success. The numerous reported recoveries admit of the probability that the procedure has also been employed in many cases which were not traumatic gastritis.

Prophylaxis. The number of cases of gastric disturbances due to foreign bodies may be considerably diminished by proper instruction of owners and attendants of animals. Harms recommends the construction of a sink-hole at the end of the crib for the reception of foreign bodies. Rust recommends for neighborhoods where hay frequently contains iron foreign bodies that the chaff cutter should be supplied with a magnet which removes iron particles. Also the installation of a blower (similar to those in threshing machines) below the chaff cutter would help to eliminate iron foreign bodies, since the lighter chaff particles will be blown farther away, while the relatively heavy iron foreign bodies will fall directly to the ground. The swallowing of hair and wool may be prevented by curing the habit of greedy licking or the itching skin affections, by constant supervision of the animals, and by separating the lambs from the ewes.

Other Inflammatory Diseases of the Fore-stomachs. (Ruminitis Reticulitis, Omasitis.) Aside from traumatic gastritis, primary inflammation of the fore-stomachs is very rare as, for instance, after the ingestion of hot liquids (distillers mash), irritating plants or caustic drugs, also under the influence of an external traumatic injury acting upon the fore-stomachs. Lichtenstern, who observed several cases of scalding of the omasum by scalded feed in one herd, found on postmortem a hemorrhagic inflammation of the omasum with a blackish-blue discoloration of the leaves and defects of the mucous membrane on the root of the tongue, on the pharynx and also in the esophagus. Dieckerhoff saw in three head of cattle a diphtheroid inflammation of the fore-stomachs and of the esophagus; since the diphtheritic foci looked very similar to trichophyton infection in cattle, the observer considered it possible that the affected animals might have ingested the mould by licking animals affected with this skin disease. In rare cases diphtheroid necrotic masses of roundish outlines are found in the rumen, in the reticulum or the omasum, also possibly in the abomasum, which have a diameter of 1 to 7 cm. After the shedding of the dead parts, the individual leaves of the manyplies have a fenestrated appearance or their margins become irregular in outline. The crusts have been seen up to 2 cm. thick, dry, fissured and dark gray; they sometimes possess a central area of softening. Complete healing

may occur in these cases with the formation of radiating cicatrices in the rumen and with permanent fenestration of the leaves of the omasum. The cause of these necrotic processes is probably a superficial invasion by the bacillus necrophorus. Superficial injuries may be caused also by the ingestion of sand and may lead to enzootics of the disease (Miessner).

Joest described two cases of chronic scirrhous inflammation with eosinophilia and partly with the formation of ulcers in the wall of the rumen, reticulum, the abomasum and even in the intestines.

The inflammation of the fore-stomachs is usually a secondary affection, appearing during the course of certain infectious diseases (foot-and-mouth disease, rinderpest, malignant catarrhal fever, sheep pox); also after gastric atony (see page 95) or overfilling of the rumen (see page 80). Tuberculous ulcers in the omasum have been described by Johne and Weiser.

The **symptoms** of inflammation of the fore-stomachs are very similar to those of gastric atony, but with that difference that repeated vomiting occurs; there is a diffuse tenderness of the fore-stomachs, and from the start a moderate fever. The rumen does not feel hard as in cases of atony. Appetite and rumination are suppressed partially, or often *entirely, while thirst is increased. If there is a concomitant inflammation* of the esophagus there is difficulty in deglutition.

In cases of hemorrhagic inflammation of the reticulum due to scalding, Lichtenstern observed, besides an afebrile condition, impossible *deglutition*, angina of the pharynx, foaming at the mouth, vomiting (even drugs given as a drench were thrown up), loss of appetite, rumination and peristalsis and an unfavorable course.

In the majority of cases the animals recover within eight to ten days. *But if the inflammation has penetrated more deeply, there is* diarrhea; the pulse-beat rises to from 80 to 100 and the animals succumb after two to three weeks from exhaustion or peritonitis.

The **treatment** is similar to that in atony of the fore-stomachs (see page 95); it is, however, not very promising in inflammations which penetrate more deeply or in those of the diphtheritic type.

3. Acute Catarrh of the Stomach. Catarrhus ventriculi acutus.

(*Gastricismus, Gastrosis, Status gastricus, Dyspepsia acuta, Febris gastrica.*)

With regard to the less perfected diagnosis of the various functions and diseases of the stomachs, we understand in veterinary medicine by the term catarrh of the stomach not only such symptoms as are caused by superficial acute inflammatory changes of the mucous membrane of the stomach, but also such as are due to the functional disturbances of the movements of the stomach and the secretion of the gastric juice.

Etiology. Acute catarrh of the stomach, including catarrh of the abomasum in ruminants, occurs either as a primary affection per se or as a secondary complication due to other organic

diseases or as part of the picture in the course of general diseases.

The most common cause of **primary catarrh** of the stomach lies without doubt in errors of diet, if an otherwise unimpeachable food material gets into the stomach without the proper preparation, in improper quantities or at irregular intervals. Starved animals go greedily for feed and try to satisfy their hunger rapidly; they do not masticate properly and do not mix the food sufficiently with saliva, hence the feed enters the stomach insufficiently broken up, in coarse masses and half dry. This irritates the gastric mucosa mechanically and the digestion of the food is insufficient. For these reasons dogs and swine are more often affected than horses; gastric catarrh is particularly common in animals which are fed only once a day. In cattle, however, the excessive ingestion of water, especially in summer, may cause catarrh of the abomasum.

Irregularity in feeding has likewise a detrimental effect, not only because the animals feed too greedily after starvation, but also because a stomach used to periodical function will easily become disturbed in the secretion of the gastric juice, if food is not forthcoming regularly. For this reason livery stable horses, which cannot always be fed regularly, suffer frequently from the disease.

Disturbances in mastication may indirectly cause gastric catarrh; this occurs in tenderness of the gums during the change of the teeth, in anomalies of the teeth, such as are seen frequently in horses, in wolf-teeth, in the different types of stomatitis with a profuse secretion of a subsequently decomposing saliva. All these factors may be detrimental to the gastric mucosa. Disturbances of rumination exert a harmful influence upon the function of the abomasum in ruminants.

Overloading of the stomach with large quantities of food which is otherwise unobjectionable occurs preferably in carnivora and hogs, because these animals are often too greedy in the ingestion of food. But horses likewise suffer from this disease if, after rather poor dry feed, they immediately receive larger amounts of palatable fresh feed, or if they can pasture at random in luxuriant clover or alfalfa fields, or if horses which have gotten loose in their barn can get into the feed supplies and can there eat as much as they want. In such cases the feed mash ferments and the gastric mucosa is irritated by butyric, lactic and acetic acid. In ruminants catarrh of the abomasum occurs through the same irritation. Starving for long periods likewise leads to catarrh. Andral and Gavaret have found catarrhal changes upon postmortem examination of starved dogs.

Improper condition of the feed may also lead to catarrh of the stomach, as, for instance, the ingestion of too cold or too hot food, such as hot distillers' mash, frozen potatoes or

beets, frosted grass, ice cold water. Hot medicinal administrations may have a similar effect.

Spoiled feed is still more harmful, especially if given exclusively or mixed with comparatively little good feed. Spoiled feeds are mouldy, sloppy, marsh-hay, mouldy cereals, moist decaying straw, intensely fermenting green feed, rotten potatoes and beets, decomposed rancid milk, fermenting old garbage, etc., and dirty foul stagnant water.

A catarrh of the stomach which is more or less severe may also be brought about if the feed contains acrid and irritating plants or food stuffs which are strongly seasoned or mixed with fragments of bone. Acrid and cauterizing drugs have a similar effect if given in a large single dose or if administered repeatedly in smaller doses, for instance, the drastic laxatives, tartar emetic, calomel, arsenic, acids and alkalies, etc.

Food of improper composition often leads to catarrh of the stomach in cattle, for instance, overfeeding with distillers' mash, germinated malt, beets and bran, potatoes, household garbage, factory remnants, leaves, bulbs. Exclusive feeding on straw likewise is often followed by gastric catarrh. Similar errors of diet in horses are more rarely observed, but they occur if these animals are fed with corn, rye, bran, crushed grain, and leguminosæ, without giving them time to adapt themselves to such food stuffs. Very young oats and insufficiently dried hay are usually also detrimental. Hogs suffer particularly if fed too much kitchen garbage. Foreign bodies in the feed are rarely of importance.

One must look for an infection if the gastric catarrh appears simnltaneously among several animals without any apparent cause and with febrile symptoms. This type is most commonly seen in horses; its cause, however is not as yet known.

Animal parasites may likewise cause catarrh of the stomach if present in larger numbers.

Of external factors over-exertion may bring about catarrh of the stomach, partly in consequence of congestiou of the peripheral blood vessels, partly if by reflex irritation the stomach is prevented from discharging its contents.

Sustmann describes a condition which he calls periodical want of appetite (weariness of food) which is found in saddle- and draft-horses, and occurs in several horses in the same barn at the same time or in succession, if they are obliged to undergo undue exertion, e. g., military horses when they are changed from track-work to outside work, to manœuvers and other hard exertion. Fiery young animals are affected particularly.

The different noxious factors do not always influence animals in the same way; they are frequently borne withont harm and particularly in animals which are not so well attended and consequently more hardened. This individual variability in resistance must be considered, and the same method of feed-

ing may be harmful to some individual animals and perfectly harmless to others.

Secondary acute catarrh of the stomach frequently accompanies acute infectious diseases and quite commonly constitutes their earliest manifestation. It is also seen in chronic exhausting diseases such as chronic diseases of the heart, lung, and liver, where venous congestion leads to catarrh of the stomach. Catarrh of the abomasum is a frequent complication of disease of the fore-stomachs in ruminants.

Pathogenesis. Some of the causes mentioned (over-exertion, overloading the stomach, debilitating and infectious diseases, etc.) prodnce a reflex irritation of the gastric mucous membrane by way of the sympathetic nerves or directly, which suppresses the contractions of the stomach and the secretion of the gastric juice; the appetite is diminished by irritation of the vagus endings in the stomach, while catarrhal or inflammatory changes in the mucosa do not make their appearance for a time, or not at all.

In most cases the gastric mucosa suffers mechanical, chemical or thermic irritation which produces hyperemia and more or less extensive migration of leucocytes into the tissue of the mucous membrane, in part also into the gastric cavity; there is further a discharge of an albuminous fluid into the stomach, with increased secretion of mucus and diminution or suppression of the gastric juice. At the same time the gastric movements are impeded mechanically by excessive quantities of food, by coarse, dry or very cold foodstuffs, particularly by various products of disintegration which are ingested with the food or develop in it later; these in their turn further interfere with the production of the gastric juice. The irritation of the gastric mucosa, or the distension of the walls of the stomach produce eructations and, in certain species, they irritate the vomiting center, giving rise to gagging and vomiting.

Since the foodstuffs are often masticated insufficiently, and therefore the gastric juice cannot act upon them readily, and owing to the presence, moreover, of secretory and motor-disturbances, the contents of the stomach soon disintegrate with free formation of carbonic acid, acetic, lactic, butyric acids, and various other products which irritate the mucous membrane and may even exert a toxic action. Spoiled feed contains such substances at the time when it is eaten. When the chyme has been propelled into the intestine, it is apt to cause intestinal catarrh. The transportation of the gastric contents into the intestine takes place only at very long intervals and, on this account, peristaltic motions become less as long as intestinal catarrh does not supervene.

Substances which give rise to fever (bacterial endotoxins, certain split proteins) are absorbed into the general circulation

only after the ingestion of greatly spoiled foodstuffs, or in case of excessive disintegration of the gastric contents, also in infectious catarrh of the gastric mucous membrane.

Anatomical Changes. Intense reddening, serous infiltration, loosening of the tissues of the mucosa, the presence of an abundant tenacious, glairy, sometimes more or less purulent mucus are the most marked anatomical changes. The mucosa is thrown into thick rugæ, which show reddening on their free margins, or even more extensively, and also occasional small hemorrhages and superficial erosions. These changes are most marked at the pylorus.

Symptoms. The most important symptom in catarrh of the stomach in all animals is a decrease of appetite, and often its entire suppression. Sometimes the animal greedily licks unpalatable, undigestible, bitter, repulsive materials, or they accept only certain kinds of food. Frequently the appetite is changeable; some meals are taken in a normal manner; then again there is an entire absence of appetite. Thirst is either diminished, or, on the contrary, increased, particularly if vomiting exists; some animals take only fresh, cool water, others again stale water or water contaminated by manure.

Another common symptom is dullness of the sensorium. The animals are indifferent to their surroundings, are feeble, apathic, stand or lie quietly in one place, do not like to move and rapidly tire at work.

The temperature is either quite normal, or perhaps there is a very insignificant elevation; the surface heat is distributed unequally. Cases which set in with high fever are rare, and even in these the fever subsides rapidly provided there are no complications.

The other symptoms vary more or less according to the various species of animals.

In the **horse** one frequently observes gaping, the animals lick iron and other cool objects and nibble on the walls. (These abnormal desires are probably due to abnormal stimuli upon the terminal nerve fibers of the pneumogastric nerve.) There is occasionally belching or even vomiting, especially if the feed had contained intensely irritating substances; such rare symptoms are however commonly seen more in dilatation or grave inflammation of the stomach. The intestinal movements are retarded, intestinal sonnds become less frequent, defecation is retarded, the feces often are discharged as small dry balls covered with a shiny coating and they contain undigested food particles. The amount of urine is, as a rule, diminished. Since catarrh often extends to the duodenum there is frequently an icteric discoloration of the mucosæ.

Cattle appear markedly feeble; they stand with their backs

curved, draw back from the crib, the skin is coarse, the ears are dropped and exceptionally they present the symptoms of dull abdominal pain. Detroye states that in exceptional cases *there are signs of agitation. The muzzle appears moist,* but if rubbed dry it takes a longer time until it becomes again covered with drops of moisture. The conjunctivæ are usually reddened and frequently show an icteric discoloration. The pulse is accelerated, small, in grave cases it cannot be felt at all. The secretion of milk suddenly becomes decreased to one half and becomes entirely suppressed in the further course.

Rumination is sluggish, infrequent, irregular or entirely absent. Disagreeable fetid gases smelling, according to some observers, like onions are expelled by belching. The region of the left flank appears either deeply depressed or somewhat more tense, but without being bloated; however, there is occasionally some bloating of the rumen in the later course of the disease, because somewhat more deeply penetrating catarrhs produce atony of the fore-stomachs. The motions of the rumen are somewhat less frequent, less energetic, but they are normal as soon as the rumen is full (Harms). Under the right costal arch, in the region of the abomasum, there is tenderness on pressure. Defecation occurs less frequently than under normal conditions; the feces are dry, dark in color, cylindrical, and they are alkaline in reaction (Harms), sometimes covered with a crust and containing coarse, undigested parts of feed. If extensive intestinal catarrh follows upon catarrh of the abomasum, there exists diarrhea.

In sheep and goats the symptoms are similar to those in cattle.

In **carnivora** vomiting is one of the most important symptoms and it is absent only in very mild cases. After overloading their stomachs, these animals vomit the ingested feed in large quantities mixed with saliva; after this their condition improves markedly, and they recover soon. In other cases vomiting only occurs directly after the ingestion of food and water, rarely independently of it; in these cases the vomited matter consists frequently of only tenacious, glairy slime, possibly mixed with streaks of blood; if the vomiting occurs very frequently bile is also present and the masses are tinged with green.

The tongue is coated and dry, the smell from the mouth is occasionally repulsive, thirst is increased as a rule, the region of the stomach under the left costal arch or behind the ensiform cartilage is tender to pressure. Defecation is retarded, at first dry, later on soft and fetid; on standing the urine forms a deposit of urates (sedimentum lateritium).

In **hogs** the disease takes a course similar to that in carnivora. The sick animals drop their tails straight down, burrow under their straw and drink much water.

In **rabbits** one observes, aside from the lack of appetite, abnormal desires like licking and chewing of the walls, ingestion of wet straw or manure, occasionally also vomiting. The abdomen appears somewhat bloated and is tender to pressure. Defecation is retarded, the feces are dry and dark in color, and diarrhea occurs sometimes in the further course. There is sometimes also an icteric discoloration of the conjunctivæ (Braun).

Course and Prognosis. Primary acute catarrh of the stomach usually runs its course in a few days, at the utmost in 1 to 2 weeks, and ends in complete recovery. However it is not advisable to make an absolutely favorable prognosis from the start, because catarrh may lead to gastritis and it may frequently be followed by catarrh of the intestines, which is of considerable importance in young and also in very old animals. Moreover catarrh may become chronic and consequently more obstinate.

The importance of secondary acute catarrh depends chiefly upon the nature of the underlying primary affection and it influences the latter unfavorably.

Diagnosis. Primary acute gastric catarrh can easily be recognized since the history of the case frequently furnishes sufficient data. It is however somewhat difficult to decide whether catarrh is confined exclusively to the stomach or whether it has already extended to the intestines. The signs which are of significance in the differential diagnosis will be fully considered under intestinal catarrh, but it ought to be pointed out here that gastric catarrh as a rule is accompanied by catarrh of the duodenum, which manifests itself in an icteric discoloration of the mucous membranes. Abdominal pain which can occasionally be observed points to a simultaneous catarrh of the intestines. Gastric catarrh is frequently accompanied by a similar affection of the intestines and in this case the disease develops into a gastro-intestinal catarrh, and it is then difficult to analyze the observable symptoms for differentiation. Intestinal sounds, the condition of the feces, and the presence or absence of icterus may however furnish enlightenment.

In ruminants it is often difficult to distinguish gastric catarrh from affections of the fore-stomachs, because the latter make it impossible to recognize whether the abomasum is intact or not, and affections of the fore-stomachs may be followed by disease of the abomasum. If there is localized tenderness upon pressure in the region of the former and an icteric discoloration of the conjunctivæ, if the left flank is fallen in or at least the rumen not full, and if further the movements of the rumen are not markedly diminished, catarrh of the abomasum may be assumed to exist. Sometimes a history of errors in diet will furnish valuable data.

The gastritis is distinguished from simple catarrh by the

greater intensity of the affection and its symptoms, by the great prostration, by the frequent and weak pulse; traumatic gastritis of cattle can be recognized by a distinct tenderness upon pressure in the neighborhood of the omasum.

Treatment. The first object of treatment is a proper consideration of the affected gastric mucosa in order to protect it against further injury. It is therefore advantageous, particularly in overfilling of the stomach, to starve the animals for a few days or to give them very little of an easily digestible food. Herbivora may have a little fresh green feed, bulbous plants, oat straw, thin-fluid salted meal or bran, or oat-meal, barley-meal, linseed-cakes; hogs may be given barley-corn or sago-soup, oat-meal with some fat and salt, scraped meat; valuable dogs may have meat-peptone as a broth in tablespoonful doses, somatose (1-3 teaspoonfuls rubbed up with broth); plasmon boiled and added in tablespoonful doses to soups or milk; roborat (2-4 tablespoons in feed or soup), tropon (like the former). The ingestion of water must be restricted as much as possible. In ruminants, however, abundant drinking water is frequently beneficial; to stimulate thirst the animals may be permitted to lick salt or they may receive mealy mashes which are strongly salted. It is proper to let the animals rest, while giving them moderate exercise in the open air; later, exercise and work are increased gradually. Only in this manner is it possible to relieve the periodical anorexia in horses (weariness of food) and to prevent it by avoiding over-exertion and by accustoming the animals gradually to harder exertions.

In overloading of the stomach in hogs and carnivora the use of emetics is indicated. The best drug is hydrochlorate of apomorphine (0.5:10 for small, 0.1:10 for large suckling pigs; 0.005 to 0.01 gm. for dogs; 0.002 to 0.005 gm. for cats), or veratrine (for hogs 0.003 gm.)—injected subcutaneously. Less advisable is the administration per mouth as for instance root of ipecacuanha (for hogs 2-3 gm.; for dogs 0.5-2.0 gm. or rhiz. veratri albi (0.5-2.0 or 0.05-0.2 gm.). Hogs may also be treated by a rectal injection of the decoctum veratri (2.0 to 50). Frequently the introduction of warm water or warm salt solution into the stomach will cause vomiting.

Washing out of the stomach (gastric lavage), may easily be practiced in carnivora and even in horses and young pigs and it is to be recommended in over-loading of the stomach.

In horses and cattle one should attempt to empty the stomach contents into the intestines by the administration of mild laxatives such as salts, tartar emetic with sulphate of sodium or magnesium (for horses 3.0 to 100.0 grammes; for cattle 4.0-6.0 gm. to 500-800 gm. in two doses). For smaller animals castor oil is indicated (for calves, foals, sheep, goats 50.0-200 gm.; for hogs 30-100 gm., for dogs 15.0-60 gm., for cats 5-20 gm.) or

calomel (according to Müller for dogs 0.3-0.4 gm.; for cats 0.1-0.15 gm.). If there is obstinate vomiting dogs should have cracked ice, water containing bicarbonate of sodium, or champagne, or opium (0.05-0.10 gm.) or other narcotics.

To remove digestive disturbances, hydrochloric acid is most valuable; it is best added to the drinking water (for horses and cattle 10-20 gm., sheep, calves and hogs 2-5 gm., dogs 0.2-0.5 gm. pro dosi). Very serviceable, particularly in horses, are artificial Carlsbad salt and laxative salts in general (horses up to 50-80.0 gm.; cattle 200.0-250.0 gm. in mucilaginous media) or combined with bitters (gentiana, calamus, rheum, etc.). Smaller animals may receive pepsin (0.1-0.5 gm.) perhaps in combination with hydrochloric acid (0.1-0.5 gm. in water); also the various bitter tinctures (Tr. rhei vinosa et aquaeosa,—tinct. gentianæ, tinct. chinæ composit., tinct. amara, vinum chinæ, vinum condurango 5-15.0 gm.). Orexinum tannicum may be given as an appetizer for dogs in doses of 0.1-0.4 gm. in broth.

4. Chronic Gastric Catarrh. Catarrhus ventriculi chronicus.

(*Gastritis catarrhalis chronica.*)

In a clinical sense we must temporarily classify under the *term chronic catarrh of the stomach* long-continued or permanent purely secretory or motor disturbances of the stomach without inflammatory changes in the mucous membrane.

Etiology. Primary chronic gastric catarrh can be produced by similar harmful agencies as the acute catarrh (page 124). In most cases the affection is due to continued feeding with spoiled or unsuitable foodstuffs (withered or frozen green feed and bulbous plants, very dry bran or meal, warm distillers' mash, germinated oats.) Frequent causes are diseases of the teeth (especially in horses), less frequent are chronic affections of the mouth, and over-exertion. The ingestion of sand with food or water produces chronic gastric catarrh in horses. Sand-disease in buffaloes and cattle (Marcone, Mueller, Prietsch) has been mentioned (page 109). In horses swallowing of air may produce chronic gastric catarrh inasmuch as the frequently repeated distension of the stomach diminishes the contractility of the stomach musculature and thus causes the food to remain in the stomach for unduly long periods.

Also repeated overexertion and old age may decrease the contractibility of the walls of the stomach and also create a tendency to insufficient activity of the stomach.

Secondary chronic gastric catarrh occurs through stasis of the portal system, in chronic affections of liver, heart and lungs. The disease which Graf described as edema of the abomasum,

and some of the cases reported by Harms as chronic gastro-intestinal catarrh of cattle probably were simply enteritis paratuberculosa. Chronic catarrh of the stomach is seen occasionally in chronic infectious diseases (tuberculosis, chronic swine plague, and hog erysipelas, glanders), in the course of chronic anemia and nephritis, also in the form of hypertrophic catarrh in young horses, probably after infectious nasal catarrh in horses (Zaepfel). Animal parasites also are a frequent cause, less so ulcers or tumors in the stomach.

Anatomical Changes. The mucosa of the stomach, particularly in the region of the pylorus, is covered with a turbid gray, tenacious and firmly adherent mucus, which is occasionally more like pus or mixed with blood. The mucosa itself is more or less thickened, corrugated either uniformly or only in spots; it is brownish red, especially at the apex of the rugæ, and it is slate-gray in color in advanced cases, presenting a more or less thickened and an irregularly patchy, nodular and folded surface, which irregularities can not be smoothed out (gastritis hypertrophica s. proliferans). In a horse dead from exhaustion the wall of the stomach was found to be three times the normal thickness: the mucosa of the stomach and of the duodenum was of a lardaceous appearance (Pr. Mil. Vb., 1907). More rarely the mucosa is pale and thin. In consequence of unequal regeneration of tissue the surface of the mucosa becomes peculiarly nodular with prominent polypoid excrescences, especially between the openings of the glands (gastritis prolifera s. verrucosa s. polyposa). Subsequently a dilatation of the stomach usually occurs.

Symptoms. In the **horse** chronic gastric catarrh usually manifests itself by a diminished appetite. The animals feed very little or not at all. This variability of appetite is occasionally accompanied by morbid desires, causing the animals to ingest alkaline, bitter and also completely indigestible materials.

The animals are dull and tire easily at work. If the catarrh has existed for some time, emaciation occurs, particularly if the animals have been overworked during this period. The mucosæ become pale or yellowish, the abdomen becomes drawn in, the hair coat appears lusterless and rough, the skin non-elastic and sometimes affected with eczema. In some cases the patients also show signs of abdominal pain.

The tongue appears coated, a tenacious, sticky, sweetish-smelling saliva frequently collects in the mouth. Defecation is retarded, the manure is dry and contains undigested food particles; occasionally there is also light diarrhea. The urine is scanty and forms a considerable sediment on standing.

Nervous disturbances may occasionally become manifest, especially dumb-staggers, and vertigo (vertigo abdominalis).

In **ruminants** sand disease of the stomach often leads to symptoms similar to those in the horse. In other cases of primary catarrh of the abomasum, which is comparatively rare, one sees diminished appetite, retarded mastication and morbid desires. In consequence of the subsequent development of a chronic intestinal catarrh there is occasionally diarrhea, alternating at variable intervals with constipation; after several weeks, however, diarrhea is the more promiuent symptom. The secretion of milk becomes entirely suppressed, emaciation and cachexia become more marked and the animal finally dies after the disease has lasted several weeks or months.

Chronic catarrh of **carnivora** hardly presents any other symptoms except variability of appetite and nutritive disturbances. The region of the stomach may be somewhat tender to pressure; vomiting is rare and usually occurs immediately after the ingestion of food. The patients vomit a tenacious mucus mixed with food particles.

Course and Prognosis. The course of chronic gastric catarrh depends largely upon its cause. If it has developed in connection with another chronic affection it is usually of grave importance because it cannot be easily cured and causes in itself a considerable ill effect upon the underlying condition. Only in purely secretory or motor disturbances of the stomach in which changes in the tissue have not yet occurred and which are due to faulty feeding, abnormal teeth, overexertion or windsucking can complete recovery be achieved by removal of the cause.

Diagnosis. The diagnosis of chronic gastric catarrh is difficult since the symptoms are usually not characteristic and since similar symptoms occur with or without gastric catarrh in various chronic diseases (particularly in diseases of the liver). A diagnosis of gastric catarrh may, therefore, be made after the exclusion of other diseases which lead to similar disturbances of nutrition.

Attention should be paid to preceding errors in diet, variations in appetite without apparent cause, morbid appetite; in carnivorons animals to vomiting even after moderate eating.

Reference to the general circumstances and the manner of feeding, examination of other organs and, if necessary, of the gastrointestinal contents and of the feces often make it possible to decide whether primary or secondary gastric catarrh is present.

Treatment. The proper regulation of diet is of much more importance in the treatment of chronic gastric catarrh than the administration of medicines.

Those food stuffs are best which are juicy, soft, mashy in consistency, since they remain in the stomach a comparatively

short time and are easily passed into the intestines. Herbivora do best with fresh green feed, soft hay, oats, straw not chopped too short with some oats, bran or oat-cuttings, also bulbous plants; the cereals which are more difficult to digest are less to be recommended; if they cannot be avoided, however, they ought to be superficially roasted or their flour ought to be first macerated at body temperature. In some cases preparations containing diastase, such as diastasolin or diafarin may be administered with floury suspensions. (According to Schade a horse ought to receive one-half pound of oat-flour stirred with one quart of cold water; this is then poured into two quarts of boiling water; when the starchy emulsion so prepared has cooled down to 55° C., 13 gm. diastasolin, previously stirred up in a cupful of lukewarm water, is added; the saccharification of the starch is complete in twenty to thirty minutes. In carnivora one may advantageously give meat broth with the yolk of eggs, finely chopped and salted meat of young animals, also milk-stews, etc. All conditions which prevent proper mastication must be looked after; the improvement of the condition of the teeth in older horses frequently leads to long-lasting improvement of digestion.

Drugs should be administered only as a measure to support the dietetic régime. Hydrochloric acid does very well in small doses (for large animals 10.0-15.0 gm., for small ones 0.5-1.0 gm.) added to the drinking water or given very much diluted (1:250 may be given as a draught). Their effect may be increased by the addition of pepsin (5-10 gm. for large or 0.1-1 gm. for small animals). The neutral salts are to be recommended. Artificial Carlsbad salt has a good effect; it is mixed with the food for horses (30-50.0 gm.) or given in the form of pills to ruminants (50-80 gm.) or it may be given for weeks, dissolved in water one-half to one hour before ingestion of food. In order to stimulate the secretion of the peptic glands, bitters may be added to the neutral salts (gentiana, calamus, rheum). Harms recommended in chronic gastric catarrh of cattle the continuous administration of Carlsbad salt in increasing doses daily from 50-150 grammes; the dose dissolved in a bucketful of water and given one hour before the early morning feeding. In ruminants the treatment used in atony of these stomachs appears indicated (see page 104).

In chronic catarrh of the stomach of a horse Lorscheid obtained very good results from strychnin (0.05 gm. strychninum nitricum on the first day and then increase daily by 0.01 gm. till 0.1 gm. is given, then decrease daily by 0.01 gm. till 0.05 gm. is again reached).

In carnivora it is advisable to add to the regulation of the diet the administration of hydrochloric acid (5-10 drops in 250 cc. shortly after the ingestion of food); also bitters (especially the bitter tinctures in teaspoonful doses, decoction of cortex

quiniæ, of colombo- or of condurango-root); also neutral salts, preferably Carlsbad salt (one teaspoonful daily in warm water upon an empty stomach). If there is much fermentation, salicylic acid (0.2-0.5 gm. before feeding), resorcin (0.1-0.2 gm.), creosote (0.1 gm.), naphthalin (0.1-0.3 gm.) in several repeated doses may be of advantage.

Gastritis Hyperplastica of Horses. Under this designation French veterinarians have described a seemingly frequently occurring and peculiar form of catarrh of the stomach.

Anatomical Changes. The stomach is either dilated or constricted, and the mucous membrane shows here and there or over extended areas nodular, warty changes which often also resemble shagreen or crocodile leather, while in other cases thick folds 4 to 5 cm. high, studded here and there with small depressions like pustules with depressed centers, are found. Microscopic examination reveals the disappearance of the pepsin glands, the greater number of which have been changed to mucous glands, leucocytic infiltration beneath the glandular layer, and sometimes the presence of **Strongylus axei.**

Symptoms. In spite of good or even increased appetite, emaciation with an increase of circumference of the abdomen will be observed, or again the appetite is depressed and the abdomen tucked up. In either case colicky symptoms, temporary diarrhea or constipation and vertigo may sometimes be observed. Attacks of salivation with sialophagia (frequent swallowing of saliva), during which the animals make licking movements with the tongue up to 100 times per minute, at other times sucking movements with somewhat protruded or retracted tongue, empty chewing, or yawning, are observed. Such attacks can also be produced artificially by tying the horse to a manger that has been thoroughly cleansed of all feed, in which case the horse will lick on the bottom of the manger for hours as though he were hastily taking up oats.

Treatment. This is successful when the dialytic process of Hayem is used, as follows: In relatively fresh cases, Solution I (sodium bicarbonate 2½, Glauber's salt 3.0, sodium chloride 1.0, water 1000); in old cases, Solution II (Glauber's salt 3.0, sodium chloride 5.0, water 1000); and in cases in which the intestinal functions are predominantly impaired, Solution IV (Glauber's salt 5.0, sodium chloride 3.0, water 1000). Three liters of the solution should be given lukewarm every morning at least one hour before feeding and during a period of one month at the most. According to the author's own observations, the solution was, in most cases, taken voluntarily by the horses immediately or after a short time and was also very beneficial in other forms of catarrh of the stomach.

5. Acute Dilatation of the Stomach. Dilatatio ventriculi acuta.

(a) Acute Dilatation of the Stomach of the Horse.

(*Magenüberladung, Magenüberfüllung, Ueberfütterungs-Kolik* [German]; *Colica crapulosa; Indigestion stomacale* [French].)

Acute dilatation of the stomach of the horse is either caused by the ingestion of unusually large masses of food, or by an

impediment to the emptying of the stomach. The heretofore normal stomach becomes dilated, but if the cause is removed it again assumes its normal volume.

Occurrence. This disease, known as overloading of the stomach, overfilling of the stomach or overfeeding colic, occurs particularly in horses which receive improper food or are fed at irregular intervals, or which are exposed to excessive exertion. This explains why the disease occurs so frequently in large cities, where the attendance, the feeding, the working of the horses is improper in many respects. The disease is rare in horses kept as breeding animals, in those kept for pleasure, or in farm animals. The affection also occurs among army horses, especially during camping and maneuvers, on account of the prevailing irregularity in feeding.

In the clinic at Budapest acute dilatation of the stomach is seen annually in 11.21% of the horses sick with colic; this is a percentage probably not markedly different from that in other large cities. The frequency of the disease varies with the season, since it is twice as prevalent during the second and third quarter as during the other quarters of the year. Most cases are seen in July and August.

Etiology. Primary acute dilatation of the stomach may be caused especially by the ingestion of unusually large amounts of feed (ten quarts or more of oats or other feed). The stomach cannot keep in motion and intimately mix an excessively large mass of feed with the proper amount of gastric juice, hence the feed remaining longer in the stomach swells and leads to the formation of large amounts of gas. The disease also occasionally appears after the ingestion of fresh hay or aftermath, or after pasturing on luxuriant pastures.

In the larger number of the cases the cause lies, however, not in the ingestion of too large amounts of feed, but in feed of improper quality; we particularly must mention in this respect indigestible corn, corn-ears, rye, marshy hay, coarse straw, feed which swells easily (bran, peas, beans) or easily form conglomerations (short-cut chaff with corn or barley-meal or bran) or food which ferments easily (young, withered clover, alfalfa, molasses, esparsette, beets and potatoes, grass heated by fermentation and green feed in general, especially if the animals drink much water after its ingestion.) Spoiled feed (moist, mildewy oats or hay or rough feed, mash) may have a similar effect. Improper food is particularly dangerous when the horse has no chance to become used to it gradually, that is, if there is a very sudden change of feed. Improper food is the more liable to produce dilatation of the stomach the more of it is ingested. The affection develops exceptionally in connection with the ingestion of too cold water (Mouquet), also with swallowed air (Goldbeck).

Overexertion or heavy work immediately after feeding also plays an important rôle in the production of the disease. Even proper and unobjectionable feed may cause dilatation of the

stomach if the horse is used for heavy work immediately after feeding. This fact, known from actual observation, has been explained by the experiments of Tangl and Scheune which show that physical exercise prevents the emptying of the horse's stomach and leads to a brisk excretion of water into the stomach, probably due to the fact that during undisturbed digestion the stomach contents become strongly hypertonic by the increase of the small molecular cleavage products, thus setting up a strong diffusion into the stomach. (This condition seems to be specific for the horse, since Jaros has found in dogs a reverse influence of the movements of the body upon the activity of the stomach.)

Certain anomalies of the teeth, which prevent the breaking up of the food stuffs, without however causing a diminution of ingestion, sometimes also cause the trouble.

The fact that some horses are more predisposed to the affection than others may be explained by a variability in the functional capacity of the gastric muscularis.

Dilatation of the stomach is frequently seen as a **secondary affection** to certain intestinal diseases. Obstruction of the small intestines leads the more rapidly to dilatation of the stomach the nearer the obstruction is to the stomach. Not uncommonly dilatation of the stomach is secondary to displacements, obstipation and bloating of the large intestine. The causes of such secondary dilatations vary. Antiperistalsis occurring on various occasions may force the contents of the intestines towards the stomach; a serious affection of the intestines also prevents the evacuation of the stomach by reflex irritation; besides every somewhat intense increase in the size of the stomach-like dilatation of the colon prevents mechanically the flow of the gastric contents towards the small intestines, because the pylorus is pressed against the stomach by the dilated and tense large intestine. Secondary gastric dilatation occurs more easily the sooner the causative affection sets in after the ingestion of food.

Pathogenesis. The causes of acute dilatation of the stomach bring about swelling of the food contained in the stomach. The gastric contents then ferment in consequence of insufficient mixing or of inherent fermentability so that more or less gas is formed. The gastric mucosa is stimulated to an increased secretion and all of these factors lead to a stretching and dilatation of the stomach. Whether after ingestion of coarse or of poorly masticated food a tonic contraction of the pylorus occurs by reflex irritation has not been proven, but neither has its occurrence been excluded. The rapidly developing tension of the gastric wall and the irritation of the mucosa by the decomposition products of feed mash produce convulsive contractions of the gastric muscularis; these cause colicky pains, which are quite intense since the contractions are severe, recur frequently and last a

long time. They are, however, *not intensified by a sudden increase* of intraabdominal pressure as in throwing down or rolling. The drier the contents of the stomach, the less gases or irritating decomposition products are formed and the less energetic are the contractions of the stomach; these muscular contractions may not appear at all after the ingestion of cold water, which acts as a paralyzant to the muscularis. The stomach pushes the diaphragm forward proportionately to its size, making respiration more difficult; however, in consequence of the diminution in the intra-thoracic negative pressure and on account of the mechanical effect against the diastole of the heart, this organ receives less venous blood, and the heart beat usually becomes more rapid. In dilatation of a high degree the internal tension can become so great that the wall of the stomach ruptures. Considerable losses of substance of the gastric wall (caused by the presence of gastrophilus larvæ, gastric ulcers) predispose to this accident. However, rupture is probably more *frequently due to a sudden increase in the intraabdominal* pressure, as it occurs in throwing down, in reckless rolling and vomiting. It can also well be imagined that occasionally rupture may occur in consequence of violent contractions of the gastric muscularis with an intense increase of intragastric pressure.

Much more frequently a rupture of the excessively distended stomach is brought about by a very sudden increase in the intraabdominal pressure as it occurs in casting or during reckless rolling. There occurs within the stomach a sudden increase in pressure which the walls of the stomach can not withstand, because it is surrounded by partly gas-containing and therefore *compressible intestines and is not protected in these* places against suddenly increased pressure. A spontaneous rupture of the stomach as a result of excessive contraction of its muscles can also be imagined and probably is also possible in the case of increased, normal, or even decreased fill of the stomach, although under such circumstances a reckless casting may play the deciding rôle by excessively increasing the pressure in the stomach, as occurs in the less frequent traumatic rupture due to the application of external force upon the region of the stomach.

Dassonville and Brocq-Rousseau experimented on the normal stomach of dead horses and found that the introduction of 26-40 liters of air or of 10-17 liters of water were sufficient to cause rupture of the gastric wall; a manometer connected with the pylorus showed a pressure of 5 cm. mercury only. Fayet and Gattend found an interior pressure of 1/12 atmosphere. Marek made his experiments by connecting the manometer with the pylorus by means of a rubber tube filled with water and he found that the normal gastric wall of the dead horse required an interior pressure of 10 to 13.5 cm. of mercury before rupture occurred.

Anatomical Changes. The stomach is dilated to a multiple of its volume; after a cut into the tense gastric wall large amounts of gases escape often in a forcible manner. In rare cases the stomach contains only food-mash and gases; usually there is much fluid mixed with the food-mash. The mucosa

shows evidence of an acute catarrh, sometimes also hemorrhages. In rupture of the stomach (ruptura ventriculi) one finds usually gastric contents, more or less mixed with blood, in the anterior portion of the abdominal cavity or between the fold of the omentum majus; the peritoneum shows the signs of an acute inflammation. Rupture occurs always in the region of the large curvature (in the experiments of the authors, mentioned above, the rupture was likewise in the same region); the margins of the tear are swollen, ragged, covered with blood coagula, the opening is largest in the peritoneal coat, smaller in the retracted muscularis, smallest in the mucosa. Sometimes the peritoneal covering of the stomach alone ruptures, while the two other strata of the wall remain intact. In other cases muscle fibers in the neighborhood of the cardiac end are pushed apart so that the mucosa here rests directly upon the peritoneum.

The lungs are in a condition of passive congestion; sometimes there is a tear into the diaphragm with a protrusion of parts of the intestines or liver into the thoracic cavity.

Symptoms. The symptoms of primary acute dilatation of the stomach usually occur within four, exceptionally only seven hours, after ingestion of feed. The patients draw back from the crib, lie back in their halter, and are listless. Very soon the symptoms of colic appear; these are severe and protracted or they may be interrupted by short, painless intervals. Sometimes such intervals are entirely absent. The animals throw themselves recklessly, roll persistently and often sit down on their haunches in dog-position. However, occasionally the colicky symptoms are only mild; they are entirely absent in dilatation of the stomach due to the ingestion of cold water (Mouquet).

The exhaled air frequently has a peculiar sour smell. Belching occurs in the majority of cases, sometimes also gagging and vomiting, with the expulsion of a sour-smelling fluid mixed with particles of food from the anterior nares and also sometimes from the mouth. One can also observe undulatory motions towards the head along the jugular groove. Pressure upon the left jugular groove often excites belching.

The stomach catheter often meets no opposition in introduction, because the esophageal muscles are relaxed. The stomach tube removes abundant sour-smelling, sometimes fetid gases and frequently also a rust-brown fluid escapes in a strong stream, particularly after lowering the head.

The circumference of the abdomen is only rarely increased. The intestinal sounds can be heard less frequently, because no food mash gets from the stomach into the intestines as long as the affection lasts; in severe cases the intestinal sounds are suppressed entirely. Cases complicated by catarrh of the intestines or moderate intestinal meteorism are char-

acterized by more intense intestinal sounds. Abdominal pain causes tenesmus and attempts at defecation by reflex irritation. However, since peristalsis is insufficient the animals have a fairly normal defecation only in the beginning of the disease, later on they succeed only occasionally in their attempts at defecation, or constipation becomes complete. Exploration per rectum usually reveals a moderate, exceptionally also an excessive degree of bloating of the small intestines, especially of the duodenum (this part is palpable immediately behind the anterior root of the mesentery, where the duodenum turns over to the left side). The spleen is frequently pushed back, so that its posterior border is displaced towards the region of the external angle of the os ileum. However, it is a mistake to attribute a great diagnostic importance to this change of position of the spleen, as is done by Forssell. For unknown reasons the base of the spleen may become displaced towards the vertical plane of the external angle of the os ileum in health, even in horses which have been starved for days. In small horses one can feel the blind sac of the dilated stomach in front of the left kidney and the base of the spleen either as a tense elastic or as a more or less firm rounded globular body which moves synchronously with the respiratory movements of the diaphragm.

The respiration becomes forced after a short time, a symptom which is markedly in contrast to the almost normal circumference of the abdomen. The pulse rapidly becomes accelerated so that after a few hours the number of pulse beats is over sixty per minute; at the same time its strength becomes diminished; exceptionally the pulse is not more frequent than normally, but it is always weaker. One generally sees an increased filling of the smaller, occasionally also of the larger veins, together with cooling of the peripheral surfaces of the body, cyanosis and perspiration. The temperature usually remains below 39° C.; but in cases complicated with inflammation of the stomach high fever prevails. E. Bauer demonstrated an increase of indican in the urine in primary dilatation of the stomach.

Although the clinical picture of secondary dilatation of the stomach is usually more or less clouded by the symptoms of the underlying primary disease, a diagnosis may possibly be made on the basis of belching, gagging and vomiting and with the aid of an exploration by means of the catheter. The stomach tube should, therefore, be introduced in all doubtful cases.

Rogers often observed in gastric colics symptoms of vagotonia (slowing down and often intermission in the pulse while applying the ear twitch) with a simultaneous increase in the reflex of the mastoid muscle upon lightly tapping it near the point of the shoulder. He tried to explain the phenomenon by an irritation of the nucleus of the vagus nerve and radiation of the excitation to the nucleus of the nervus accessorius. In a similar manner he thought it permissible to assume an increase in the reflex irritability of the skin of the withers, the shoulder and the abdominal wall, due to painful affection of the abdominal organs whose nerve connection with the spinal cord lies in the same section of the cord as the reflex arc in question (radiation of the increased nervous irritation).

Course and Prognosis. The symptoms usually subside in mild cases within a few hours and disappear completely after a few more hours; the symptoms of acute catarrh of the stomach, however, not infrequently persist for a few days, and an accelerated and weak pulse may be observable for one or two days. In some cases immediately after the animals appear to have recovered, the symptoms of acute gastric inflammation appears, and they die from it. Pulmonary gangrene develops not uncommonly within a few days, and is due to aspiration after belching or vomiting.

In grave cases (after the observations of the authors in 15 to 33%), the symptoms of gastric dilatation increase in intensity and the patients die with manifestations of great restlessness or after they have already quieted down somewhat. If the patients are treated early with the stomach sound they can usually be saved. Among the causes of death may also be enumerated suffocation, rupture of the stomach, rarely rupture of the diaphragm. The muscularis of the excessively dilated stomach may lose its contractility entirely, so that the pains cease almost completely; but the forced respiration, the very weak pulse and the anxious expression announce the impending suffocation.

After **complete rupture of the stomach** the colicky pains suddenly cease, but the general condition rapidly becomes worse. The animal becomes prostrated, shows extensive muscular tremors, staggers and is bathed in perspiration; in spite of quieting down it does not ingest food (in two cases of Sequens the appetite for food and thirst was retained in spite of rupture); the eyes become staring, the pulse beat rises above 100; but it cannot be felt. Tenderness of the abdominal wall can hardly be demonstrated. If an exploratory puncture is made in the lower part of the abdomen, one can usually obtain a sour or neutral fluid which is hemorrhagic and mixed with particles of feed, particularly grains (Fig. 10). Rectal exploration meets no resistance in the upper portions of the abdominal cavity, because in this part the gases are collected which have escaped from the stomach. In cases which do not take a too rapid course, one can feel particles of food on the peritoneum, which is rough and tender. (Roughness of the peritoneum in itself alone is no proof of rupture of the stomach or peritoneum, because it occurs in peritonitis due to other causes.) The temperature sometimes goes below normal at the start, but rises later if the animals remain alive for any length of time.

Death after rupture may occur within a few quarters of an hour, more rarely only after several hours. If the gastric contents get between the leaves of the mesentery only, the animal may live for several days, and then the symptoms of acute peritonitis can be observed distinctly. Fiebiger saw additional symptoms of emphysema of the skin due to a tear into the parietal layer of the peritoneum.

Pecus claims that rupture of the stomach is announced by a peculiar sharp neighing with subsequent vomiting and the expulsion of the gastric contents through the nose. (It is probable that reference is here made to the peculiar squeaking sound not infrequently heard in horses during a convulsive contraction of the abdominal walls in vomiting; but rupture of the stomach does not necessarily follow the former.)

Rupture of the diaphragm is announced by the symptoms of collapse and increased difficulty of respiration, also by a tympanitic or metallic sound on percussion, which changes from

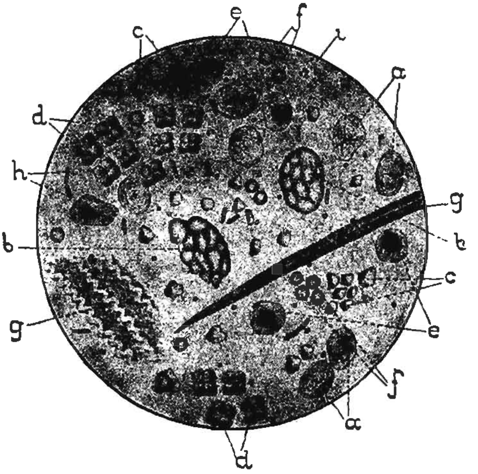


Fig. 10. *Sediment of the intra-abdominal fluid in rupture of the stomach.* *a*, large, *c*. small granules of starch of cereals and leguminosæ; *b*. starch granules of oats; *d*. sarcinæ; *e*. white, *f*. red, blood corpuscles, the size of which varies very much according to the concentration of the gastric contents; *g*. plant fibers; *h*. bacilli; *i*. cocci.

time to time, but is usually found over the left side of the thorax; or, on the contrary, by a circumscribed dullness behind the cardiac region. Exceptionally the diaphragm is pushed forward by the much dilated stomach; the latter together with the diaphragm then comes in contact with the thoracic wall and a tympanitic sound is heard behind the cardiac region in spite of the fact that there is no rupture of the diaphragm. Death generally follows soon, but there are exceptional cases; the animal remains alive, a diaphragmatic hernia forms and later on this gives rise to recurrent attacks of disease.

Diagnosis. The most important signs for the recognition

of acute dilatation of the stomach are the appearance of colicky symptoms after the ingestion of large masses of improper food or after the horse has done work directly after feeding, severe and persisting colicky pains not accompanied by any marked bloating, early appearance of a weak pulse and of difficult respiration, moderate bloating of the small intestines, belching, gagging and vomiting and finally the result of the examination with the stomach tube. Belching, gagging and vomiting or the evacuation of masses of gases and fluid through the stomach tube are also observed in secondary dilatation of the stomach; on the other hand, belching, gagging or vomiting may be absent in primary or secondary dilatation of the stomach. It appears always advisable to examine the abdominal organs by a rectal exploration, because other affections, accompanied by abdominal pains, and similar symptoms, may appear shortly after the ingestion of food. For differential diagnosis are particularly to be considered displacements of the intestines (twisting, volvulus, invagination, internal strangulation), grave forms of thrombosis and embolism and other possible causes of colicky affections.

Treatment. The main object of treatment is the evacuation of the contents of the stomach. Mild cases of primary dilatation of the stomach may be treated by laxatives, especially aloes (25.0-35.0 gm.) alone or with neutral salts (100-150 gm.) or rheum (20-25.0 gm.), or salts alone in large doses (sulphate of sodium, sulphate of magnesium) (200-300 gm.) dissolved in much water, as concentrated solutions of the neutral salts are apt to inhibit the contractions of the stomach. Forssell recommends for the affection 12 gm. of lactic acid (75%) in a pint of water, using it with the apparatus of Goldbech; in dangerous cases a repetition of the dose may be indicated after one-half to three-quarters of an hour. Forssell claims that the administration of lactic acid imparts to the gastric contents that degree of acidity which will favor its removal into the intestines. The experience of the authors, however, teaches that grave cases of gastric dilatation cannot be cured by this treatment and that evacuation by the stomach tube becomes necessary in order to avoid rupture of the stomach or suffocation. One may, on the other hand, inject subcutaneously sulphate of eserine (0.08-0.10 gm.) or arecoline (0.06-0.08 gm.) or hydrochloride of pilocarpine (0.15-0.25 gm.). In cases with considerable elimination of water, arecoline and pilocarpine are not entirely harmless on account of their action by which they increase the secretion of saliva and of the gastric juice. These drugs have to be used very cautiously, because they may cause sudden death by paralysis of the myocardium or strong contraction of vessels in animals suffering from diseases of the heart or lungs, or they may increase the contraction of the gastric muscularis, increase the pains, cause more reckless rolling and so increase the danger

of rupture of the stomach if its contents are not expelled, in spite of their administration. In order to reduce the pain, and to slow up the action of these remedies, which is usually very rapid, Schadrin advocates a preliminary injection of morphine (0.4-0.6 gm.). Less dangerous are fractional doses of eserin (0.02 to 0.03 gm.), pilocarpin (0.05 to 0.08 gm.), or arecolin (0.01 to 0.02 gm.) at intervals of 1 to 2 hours.

• Chloride of barium should never be used as in other colicky affections of the horse, because occasionally it causes sudden death or a rupture of the stomach. During those years when chloride of barium was used extensively in the Budapest Clinic on horses with colicky affections, fatal results occurred in one third of the cases, due to rupture of the stomach intra vitam. Hence this drug is used no longer in horses with colics in the Budapest Clinic and the mortality is less than it was formerly.

The attempt should be made to quiet the great unrest by subcutaneous injections of morphine (0.3-0.6 gm.) or by internal doses of chloral hydrate in gruel (Joyeux); the latter drug is given better per rectum. In place of the intraperitoneal injection of 25 to 30 gms. chloral hydrate in 10 parts of 1 per cent saline (Breton), which is not without danger, it is more advisable to give intravenous injections of the drug, on the average 50 cc. of a 7½% solution, whereupon the animal will lie down voluntarily and remain unconscious for a long time (Caemmerer). Opium is far more appropriate and Bambauer claims that it may be successful even in desperate cases (120-200 gm. of the tincture, by mouth). The throwing and rolling of the animals is to be prevented by watching. In view of the fact that motion prevents the propulsion of the gastric contents, the sick animals should not be made to take exercise.

Laxatives are of no avail in grave cases, and in these, as indeed in all cases of gastric dilatation, the stomach should be emptied with the aid of the stomach tube and should subsequently be irrigated. This is the only rational treatment which can be carried out not only in the clinic, but also in private practice. After the gases and fluids have left the stomach and after the latter has been irrigated, the animals at once become quiet, but the dilatation has been complicated by intestinal catarrh or intestinal meteorism. As a rule only one evacuation of the stomach is necessary, but occasionally the operation must be repeated. If eructations occur, a single or, if necessary, repeated introduction of the ordinary stomach tube or of a whipstock reversed, make the discharge of the stomach gases possible, according to Bambauer.

Evacuation of the gastric contents by the stomach catheter. The latter consists of a tube 2.25 meter long, 27 mm. in diameter of ordinary red rubber or of the more economical "durit"; its inner diameter is 16 mm. The tube is armed with a flexible rod (cane) of the proper strength and the rod is provided with a conical button at its distal end. (Fig. 11.) This stomach catheter constructed by Marek has the advantage over Monroe's ordinary stomach tube, that it is less easily damaged by the teeth, and that it will remain in good condition for years if protected against the action of fats; however it becomes longer and somewhat thicker if used frequently.

The introduction of this catheter is possible without difficulty in almost every horse with the proper assistance (at least 3 to 4 assistants are necessary); with the animal standing up, the introduction is frequently as easy as in cattle. Two or if necessary more assistants draw the head of the animal down and introduce a Bayer mouth-speculum between its molars or they introduce a mouth gag wrapped in cotton, oakum, etc. The assistant who holds this instrument elevates the lower portion of the head in a manner to bring it in a line with the neck. The tongue is now drawn laterally out of the mouth, the catheter end to be introduced is made slippery with oil, fat, vaselin or best with glycerine, and is now carefully, but rapidly introduced with both hands along the hard palate into the pharynx and pushed along until the introduced portion is equal to the previously measured distance between the cardiac end of the stomach and the incisors. After the introduction of the catheter rattling generally occurs, but this is of no significance. If the catheter meets any *resistance before getting into the stomach, this may be overcome by withdrawing the* instrument several times and then pushing it along again or by removing the rod and pouring 1-2 quarts of lukewarm water into the tube; this brings about a relaxa*tion of the esophageal wall and of* the cardia. After the rod has been withdrawn the tube should be well introduced into the stomach, otherwise the procedure may not be successful. (The length of the introduced tube should be 116-125% of the height of the animal as ascertained by a tape measure.) If there is no evacuation of the stomach after removal of the rod, this may be brought about by closing the outer end of the tube during inspiration and opening it during expiration; in this manner, we make use of the suction produced by the negative intrathoracic pressure. In other cases the desired end may be obtained by pulling forward and backward on the free end of the tube or by allowing 2-4 quarts of water to run into the tube, while the head of the horse and the end of the tube projecting out of the mouth are held down before the water has all run in, producing in this manner syphon-action of the tube. Sometimes a partial removal and reintroduction of the rod may be necessary. The head of the horse must always be lowered considerably if an unimpeded *evacuation is to be assured. (The amount of evacuated gastric* contents usually varies between 15 and 30 quarts.)

Fig. 11. Introduction of the stomach catheter into the horse.

Catheterization of the stomach on the animal in a recumbent posture is almost never necessary and it does not appear to be practicable, since dyspnea may reach a high degree and since the advantage of the syphon action available on a standing animal is lacking.

Catheterization must be performed with care, otherwise fatal injuries to the wall of the esophagus or to the intrathoracic vessels may occur.

The authors cannot recommend the introduction of the stomach catheter through the nasal cavity as proposed by Phillips, because the introduction through the mouth is much easier and permits the use of a much thicker catheter.

Irrigation of the stomach becomes necessary in those cases, where the gastric contents are thick-fluid or mashy, or more or less red stained, or when a firm consistency prevents their evacuation. Irrigation of the stomach is carried out in such a manner that the free end of the tube is provided with a funnel through which 2-5 quarts of lukewarm water are allowed to flow into the stomach and are then removed in a manner already described; this procedure is repeated several times. *(In a grave case of acute gastric dilatation after the ingestion of corn* ears, such irrigation was performed 5 times in 7 hours and a total of 122 quarts of water was used.)

It is necessary in all cases to keep the patients under observation for several

hours after the evacuation of the stomach, so that the procedure can be repeated if required by renewed symptoms of colic or by an aggravation of the general condition.

The therapeutic value of the use of the stomach catheter is clearly shown by statistics. The method has for several years been used in the Budapest Clinic; of 85 horses so treated in 1906, 81 (95.3%) recovered. Behrens reported from the Berlin Clinic 93.7% recoveries and other observers reported similarly favorable figures.

After recovery the animals should be starved for one day and they should then receive easily digestible food for some time.

(b) Acute Gastric Dilatation in Other Domestic Animals.

Occurrence. Among carnivora, especially among dogs and hogs, acute dilatation of the stomach is often observed; it is rare in rabbits, and in ruminants, particularly in cattle.

Etiology. In **carnivora**, in **hogs** and also in **rabbits** acute dilatation of the stomach is caused by overloading the stomach, particularly by the ingestion of large amounts of indigestible, dry or of easily fermentable feed. Potatoes, bread, dog biscuits, blood, bran, withered green feed, leaves of beets, leguminosæ, are in this respect dangerous. (Bruckmüller found whole pigeons with large masses of claws, feathers, skin and bones in the stomach of dogs which had died of starvation in consequence [Kitt]).

In **cattle** acute dilatation of the abomasum occurs rarely as a primary affection after the accumulation of masses of dry feed in the abomasum (Harms, Lindenberg, observation of the authors) if the feed gets into the omasum or abomasum without rumination, or if it has not been broken up sufficiently, in spite of a double mastication. Exceptionally the condition may develop secondarily, for instance in the course of gastric atony (Koch, Frasch) which gives insufficiently prepared feed a chance to enter the abomasum. In both cases the coarse condition of the feed prevents the pylorus from opening and hinders the entrance of the feed mash into the intestines.

Anatomical Changes. The stomach is enlarged, its wall drumlike, or the stomach may be stuffed with dense masses of feed. In hogs and rabbits rupture of the stomach is occasionally observed at the large curvature, which has taken place intravitam (Micucci, Joest). In cattle the abomasum filled with masses of feed may be enlarged to the size of the rumen. (In a case observed by Harms the abomasum contained forty-five quarts of dried feed.) Exceptionally a rupture of the abomasum may be found either confined to the serosa and muscularis or it may be complete; in either case, it may lead to partial or general peritonitis (Harms, Lindenberg, Gückel).

Symptoms. Overloading of the stomach comparatively rarely leads to grave disturbances in **carnivora**, in **hogs** or **rab-**

bits, because these animals can vomit easily and can thus clear their stomach of excessive contents. If, however, an equilibrium cannot be brought about in this manner and if vomiting of very dry coarse feed becomes impossible, there occur gagging, salivation, bloating, first of the left side later on also of the right side, in the region of the lower ribs and finally of the whole abdomen, with a frequent pulse beat, forced respiration, anxious expression, whining, howling, groaning, sighing and restlessness. In carnivora and in rabbits the dilated stomach can be felt through the abdominal walls. Frequently symptoms of bloating of the intestines also supervene. In grave cases the patients succumb within a few quarters of an hour in consequence of rapidly increasing dyspnea, while in milder cases recovery occurs slowly or the clinical picture changes into that of acute gastric catarrh.

The wheezing of fattened hogs is caused, according to Weisz, partially by an acute gastric dilatation after the ingestion of sour bran. A few hours after feeding a forced whistling respiration in a sitting posture is observed, the animals are restless, they groan from time to time and succumb within a short period.

In **cattle** obliteration of the abomasum leads to lack of appetite, also of mastication and secretion of milk, absence of thirst, listlessness, sluggish movements. Harms alone has observed frequent belching and vomiting. The left flank may be sunken in deeply (Harms) or it may be normal or bulging (Koch, Frasch, authors' observation), but it is of normal consistency. The movements of the rumen occur somewhat less frequently; but they become normal after the administration of drugs which stimulate gastric movements, without, however, producing a change in the condition of the animal. A fairly constant symptom is tenderness of the region of the abomasum. Defecation is at first normal, but becomes less frequent and ceases entirely on the third day of the disease. At this time an increase in the number of pulse beats can be observed and also a moderate elevation of temperature. The disease always ends fatally unless the animals are slaughtered before natural death occurs.

Diagnosis. In carnivora, rabbits and hogs the affection can easily be diagnosticated upon the basis of the history and the clinical signs, and in dogs torsion of the stomach can be excluded. Primary obstruction of the abomasum in cattle is suggested by complete lack of appetite from the start, cessation of rumination and of the secretion of milk, with minor disturbances in the functions of the fore-stomachs; the impossibility of influencing the lack of appetite, rumination and defecation by proper drugs; tenderness upon pressure in the region of the

abomasum. The exclusion of acute affections of the fore-stomachs is, however, always somewhat difficult.

Treatment. The removal of the gastric contents frequently may be brought about by the administration of emetics (apomorphia, veratrine; see page 131). Washing out of the stomach may become necessary; it is easy in carnivora, and the principles of the procedure are the same as in the horse; the stomach tube which is used must, of course, be much thinner. In obstruction of the abomasum in cattle, treatment with laxatives (see page 85), has never been successful. In hogs the stomach is easily emptied by lavage if care is taken to avoid the pharangeal recess in passing the stomach tube through the pharynx. The tube easily enters this recess and if the slightest resistance is noticed, a stiff tube may pierce the pharyngeal recess, passing through the loose connective tissue of the neck into the thorax, and cause fatal pneumothorax.

6. Chronic Dilatation of the Stomach. Dilatatio ventriculi chronica.

Chronic dilatation of the stomach is an enlargement of the stomach which persists, at least for some time, even after the removal of the cause or after evacuation of the gastric contents, and which may be likened to chronic atony of the fore-stomachs of ruminants.

Etiology. Primary dilatation of the stomach frequently occurs after feeding, for a long time, non-nutritious feed, such as chaff, coarse straw, coarse leguminosæ. Such food is also detrimental because the animals are forced to ingest larger amounts in order to satisfy their requirements. Animals which are excessive feeders are in particular danger. The gastric muscularis is too weak in some individuals to propel a feed mash of this kind, consequently the food remains too long in the stomach; it becomes decomposed and in the course of time causes a chronic dilatation of the stomach. Dilatation of the stomach on this basis has been observed by Trinchera in 2-3% of young horses, mostly three to five years old, which had been imported from the provinces of Carinthia, Salzburg and the Tyrol to Italy, and it has also been observed in horses from the north of France, from Croatia and Dalmatia. Sand and gravel swallowed in large amounts exert a similar effect. (Dilatation of the stomach due to an accumulation of sand combined with an accumulation of sand in the intestines frequently occurs, according to Wedernikow, in the Steppes of Kirgisia (Siberia) and is known as Gata-disease (see page 132). Persistent swallowing of air not infrequently induces chronic dilatation of the stomach, which may be due also to the repeated occurrence of the acute dilatation, particularly in dogs.

Persistent obstruction at the pylorus (pyloric stenosis due to tumor formation or to cicatricial contraction) or in the small intestines (stenosis, frequent obstipation, intestinal calculi) usually leads to secondary dilatation of the stomach. The cause of this affection may also be found in atony of the gastric muscularis due to chronic catarrh of the stomach, to tumors of the stomach, to sclerosis of the muscularis (Liéuaux), or to chronic interstitial hepatitis of horses (socalled Schweinsberger disease); in the last named disease the ingestion of food which is difficult of digestion plays an important rôle.

Since the muscularis of the stomach becomes weakened with advancing age and since mastication likewise becomes deficient, chronic gastric dilatation is frequently seen in older animals.

Pathogenesis. The repeated overfilling of the stomach with food or with swallowed air at first leads to an enlargement of the lumeu of the stomach, by accommodation, and the more vigorous or even convulsive contractions of the stomach muscles for a time make the undisturbed propulsion of the contents possible. As the muscles constantly do excessive work, they become *hypertrophic in the course of a few weeks, and therefore* it does not appear to be thinner in spite of the increased volume of the organ. If the discharge of the contents is prevented mechanically, the lumen of the stomach remains unchanged for a time, except that the muscles become hypertrophic because they must propel the food-mash against an increased resistance. After weeks or mouths, in both forms of dilatation, the gastric muscles become lame and the emptying of the stomach is retarded. In the accumulated contents an abnormally brisk fermentation and disintegration takes place, the gases which develop continually distend the stomach and lead to, or increase dilatation, aided by the action of certain products of the disintegration, which inhibit contractions. In primary debility of the stomach muscles, the organ is emptied insufficiently from the beginning and this gives rise to a gradual increase of the lumen without a preceding hypertrophy of the muscles. In any case, the gradual increase of the dilatation finally may abolish the contractility of the stomach muscles and arrest the transportation of the gastric contents.

The dilated stomach interferes with respiration, in proportion to its size, and influences the blood circulation unfavorably, especially during the time when it is filled with food.

Anatomical Changes. The stomach is sometimes increased to several times its normal size, and in grave cases its shape may also be changed (the stomach of the horse becomes egg-shaped or similar in shape to the rumen of cattle), its wall is generally thickened, occasionally also thinned, sometimes as thin as brown paper (Fitzroy-Philipot), sometimes also fairly tough (Liénaux). The mucosa generally presents the signs of chronic

catarrh. Sometimes we find as the cause of a dilatation a tumor in the stomach, in the pylorus or in the intestines.

In a case of Koch, the stomach of a horse weighed 51 kilograms when full and had a cubic content of 84 liters (the normal cubic contents being 7 to 15 liters); its largest diameter was 164 cm. Fitzroy-Philipot found a full stomach of a horse weighing 71 kg., it weighed 10 kg. (about 20 pounds) when empty and its length (between cardia and pylorus was 261 cm., the greatest thickness 190 cm. Liénaux found the larger curvature in an adult setter dog to be 50 cm. long. However, increases of this extreme degree are seen only exceptionally.

Symptoms. **Horses** suffer in this affection frequently from repeated attacks of colic (socalled chronic or periodic colic) which are much like those seen in acute dilatation (see page 136). *Such attacks occur in stenosis of the pylorus almost regularly* towards the end of feeding or immediately after it; in other cases shortly after the meal; they frequently last for several hours. Each attack may, of course, become fatal in consequence of suffocation, rupture of the stomach, etc. In the further course of the disease the attacks of pain become less severe; however, the general condition does not improve and the attacks last a longer time; the animals suffer more and more in their general condition. Respiration is also difficult in the periods between the attacks, and some animals may develop roaring. Finally, when the gastric muscularis has lost its contractility, no more attacks of pain occur, but the patients soon succumb to exhaustion, rupture of the stomach, suffocation or peritonitis.

In other cases, when dilatation is due to catarrh of the stomach, or to improper food, one sees from the start the clinical picture of catarrh of the stomach in combination with difficult respiration, if the dilatation is at all considerable. In such cases and in those described above, the dilated stomach may be palpable from the rectum. During the slow development of the dilatation the organism has time to adapt itself to changed conditions, hence during rest the pulse does not present a marked deviation from the normal. However, *rupture of the stomach* may occur at any time.

Trinchera has described a condition under the name of "abdominal foundering of young horses," which is due to gastric dilatation after improper feeding, and which is characterized by slow and hesitating inspiration, followed by quick interrupted expiration with drawing in of the flanks and bulging of the region of the lower ribs. These disturbances are most marked during and after the ingestion of food. If proper feeding is instituted the disturbances disappear after an interval which is longer than that during which the affection has persisted.

These observations of Trinchera are in accord with others made in the province of *Salzburg and showing that the permanent ingestion of voluminous* food or the sojourn in poor pastures *is followed by similar disturbances in respiration* which however disappear on proper feeding (change of owner).

Oppenheim observed a case of chronic dilatation of the

abomasum in **cattle.** The animal vomited daily for weeks, especially at the beginning of rumination. In a case described by Koch the affection followed upon atony of the fore-stomachs and its symptoms were obscured by those of the atonic condition of the latter.

In **dogs** the symptoms are those of chronic gastric catarrh. The filled stomach may be palpated in both regions of the lower ribs; if its space is not completely filled with masses of food, percussion shows a tympanitic sound over a space between the eighth rib and the umbilicus (Schindelka); this sound becomes weaker or disappears after the ingestion of food; sudden pressure upon the gastric region elicits splashing sounds.

Liénaux observed dilatation of the stomach of a dog, which appeared simultaneously with a sclerosis of the muscularis. The affection at first came on in periodic attacks, it then became permanent and led to diarrhea, which resisted every treatment, the feces contained almost unchanged food, particularly meat. The appetite was good. Behind the diaphragm one could feel a large immobile mass. The absence of vomiting was believed to be due to the stiffness of the stomach wall.

In a dog with infrapapillary duodenal obstruction by a foreign body, Dasch observed motor insufficiency of the stomach with simultaneous tetany. In the gastric contents which were removed by stomach pump, after a test meal, free hydrochloric acid could be demonstrated only after the walls of the stomach had been thoroughly flushed with water, whereby the secretion of gastric juice was stimulated.

Treatment. If treatment is at all desirable, the ingestion of easily digestible, or still better, fluid feed (see page 131) may retard the development of fatal symptoms and the general condition of the animals may be preserved to a certain degree.

To remove the accumulated gastric contents the same means may be employed as recommended for gastric atony or for acute dilatation of the stomach (see pages 104 and 144). Pyloric stenosis in dogs may be treated by a gastroenterostomy as practiced in human surgery; this operation has often been performed successfully in experiments on dogs.

7. Foreign Bodies in the Stomach. Corpora aliena ventriculi.

Etiology. Aside from what occurs in rabies, in playing, retrieving or also in devouring their food, dogs frequently swallow foreign bodies, such as, stones, pieces of money, corks, bits of glass, skeins of twine, pieces of cartilage, bone, rags, sponges, needles, etc.; hair balls are rarely found in the stomach of dogs.

In cats, pieces of mice or rats, or whole mice, are sometimes arrested in the stomach, other foreign bodies only very rarely; such foreign bodies may be needles, fruit seeds, etc.

Pathogenesis. As long as foreign bodies remain free in the stomach, they usually do not produce any harm, but according

to their nature, they frequently lead to acute or chronic catarrh or inflammation of the stomach. Sharp or pointed bodies frequently injure the gastric mucosa and penetrate it, leading then to perforation or fistula. Pointed as well as dull bodies may become wedged in the pylorus and may bring about its complete obstruction.

Swallowed pins rarely cause gastro-intestinal disturbances. Zoja, who experimented on cats, made them swallow 125 sewing pins: only two penetrated, one above the pylorus, one into the rectum; the others were either voided through the anus or were found in the killed animals in the large intestine where they had not led to any injury (Eichhorst).

Symptoms. These occasionally are similar to those of an acute, more rarely of a chronic gastric catarrh; they are more frequently similar to those of inflammation of the stomach: the lodgment of the foreign body in the pylorus produces the symptoms of obstruction of the pylorus. In the latter case the following symptoms are observed: Obstinate vomiting, lack of appetite, sometimes intense thirst, excitement, frequent change of posture, loud howling, groaning, inclination to bite, convulsions, finally complete listlessness. The stomach is tender to pressure and the foreign body may occasionally be felt in the region of the lower ribs behind the liver (Udriski felt the end of a hairpin in a cat). The weakness rapidly increases, fever sets in, the pulse becomes rapid and weak and the animal succumbs.

In a dachshund which while retrieving swallowed 180 pieces of gravel with a total weight of more than 1 kg., Jakob and P. de Jong observed on the following day only a decreased appetite and slight depression, and on the third day the usual symptoms. The lower wall of the abdomen appeared bulged out like a barrel; during palpation a large number of stones could be easily felt, and on percussion a plain rattling of the stones could be heard over the entire region of the greatly distended stomach.

In Coquot's case in a fox terrier 7 stones (largest the size of a hen's egg) with a total weight of 460 gm. had been swallowed. On palpation of the region of the stomach, one had the impression of pressing upon a sack filled with nuts.

Hinz detected in a dog that had swallowed a steel ball weighing 73 gm. and a stone weighing 40 gm., when the animal was raised up in front and the abdomen lightly tapped at a point 3 fingers wide posterior to the xiphoid cartilage and a little to the left of the linea alba, a distinct sound as though stone and steel were tapped together. He diagnosed from this a diverticulum per pulsionem ortum produced by foreign bodies. Similar sound phenomena were observed by Wilson.

Diagnosis. On the basis of the symptoms described and in connection with the history of the case a diagnosis can, as a rule, be secured. Occasionally one might resort to an exploration of the abdominal cavity by the aid of Roentgen rays. However, it must not be forgotten that a large portion of the stomach in a Roentgenogram is covered by the liver, hence bodies which have not a high specific gravity may not become visible. It is advisable when making an X Ray plate to have the animals on their hind legs, so as to bring the pylorus below the liver. The stomach might also first be filled with carbon-dioxide gas.

Treatment. The removal of a foreign body which is neither sharp nor pointed may be attempted by an appropriate emetic (apomorphine 0.005-0.01 gm.). If this treatment is not successful, or if it cannot be tried on account of the nature of the foreign body, laparogastrotomy is indicated. With its aid Hamoir, Vennerholm, Udriski and Bergeon have saved dogs and one cat.

Jakob and de Jong saw recovery in the above mentioned case in 17 days simply by generous feeding and frequent rectal infusions of olive oil. In Frontori's case recovery took place in a short time after a brooch 12 cm. long, having been swallowed with a piece of meat, had penetrated through the skin in the left hypochondrial region and a piece 8 cm. long had been cut off while the rest was pushed back into the stomach. A similar course was observed in Seidler's case in a dog that had swallowed a hypodermic needle, which, with its pointed end, had penetrated through the skin behind the last rib, and after being drawn out as far as possible was cut off with a pair of pliers.

Foreign Bodies in the Stomach of Other Animals. Foreign bodies are only very exceptionally found in the stomach of the **horse.** Hahn and Marek each found a piece of wood, Eberbach and Enke needles, respectively, 21 and 6 cm. long, Wölker and Grundmann found pointed fragments of bone in the stomach or its neighborhood in the cadavers of horses which came to post-mortem examination. Foreign bodies cause inflammation of the stomach, perforation of this organ, or inflammation of the neighboring organs and of the peritoneum. Schmidt saw traumatic inflammation of the stomach, diaphragm and lungs of a horse. In a few cases feed-balls (Bayer) or gastroliths were found on post-mortem examination in horses which had died with symptoms of colic (in a case of Moebius the stone found in the stomach weighed 324 gm.; in a case of Johne 750 gm.).

In the stomach of **swine** *foreign bodies occur somewhat more frequently* (pointed objects, pieces of wood, hair balls), with the exception of hair balls, these usually lead to perforation of the stomach and to secondary peritonitis.

Brown found two hair balls in a **rabbit.** The animal swallowed its own hairs as it licked them off, and they were glued together by the gastric mucus.

In **fowl** foreign bodies are not infrequent. The animals take them up deliberately or accidentally while ravenously picking up feed (especially grain) from the ground, as well as while habitually ingesting sand and small gravel for the assistance of digestion, whereby pieces of wire, needles, nails, pointed pieces of glass and other foreign bodies are easily swallowed (Eber), especially when the birds are fed in flocks or are poorly fed and prevented from taking exercise in the open. The foreign bodies remain in the crop and in the glandular stomach for a while, but then reach the muscular stomach (see Plate II) and occlude its opening. Pointed and cutting foreign bodies injure the wall of the stomach and may perforate it, especially in geese, ducks and pigeons, most often near the beginning of the intestine.

According to Schantyr's observations, foreign bodies which are only partly imbedded in the wall of the stomach will, in about 5 days, become surrounded by necrotic masses. In case the foreign body is of

iron it is slowly destroyed by oxidation, whereupon, after the regeneration of the tissues, total recovery takes place. Foreign bodies which penetrate the wall of the stomach for their entire length may remain in the perforated part for a long time without causing changes in the musculature of the stomach. They become blackened as a result of the oxidation of the iron. In some such cases, however, a complete perforation may develop later. Following complete perforation the foreign body at times falls directly into the abdominal cavity; as a rule, however, it remains partly lodged in the wall of the stomach, not infrequently leading to a fatal peritonitis caused by microorganisms that have been carried along and by stomach contents that have gradually oozed out. Much more frequently only a circumscribed gastroperitonitis is produced, whereby fibrinous exudate is formed around the projecting foreign body, which is later displaced by a connective-tissue capsule forming adhesions between the stomach and the abdominal wall or other abdominal organs, at times also with the pericardial sac. The adhesions frequently contain abscesses of a black coloration containing thick pus. At this stage of development the process may become checked, after a dissolution of the foreign body, without causing any bad effects; as a rule, however, a general peritonitis or an inflammation of the air sacs will sooner or later develop. It may even lead to a perforation of the abdominal wall with the formation of a fistula or of a cavity containing decomposed stomach contents (Joest).

The symptoms consist in a gradual diminution of the appetite, constipation and rapid emaciation increasing to cachexia to which the animals succumb. If perforating foreign bodies are present, the stomach, partially enclosed in masses of fibrin, is felt as a rounded tumor in the abdominal cavity not very elastic and sometimes not movable, and it may be mistaken for an arrested egg in female animals. In the case of Joest which has been referred to, the pigeon showed disturbances in respiration, and died suddenly when the attempt was made to grasp it.

According to Hébrant & Antoine the only proper treatment is laparotomy with removal of the foreign body, together with its fibrin capsule, after which the abdominal wound heals readily. If the perforation is incomplete, the foreign body is withdrawn from the stomach and removed, also with the fibrinous exudate; the stomach wound is then sewn up in like manner as after gastrotomy for the removal of blunt foreign bodies which occlude the opening.

According to Schantyr the operative removal of the foreign body by laparotomy should be reserved for cases where a complete perforation has taken place, since gastrotomy always leads to a general inflammation. Even in complete perforation it is advisable to abstain from the removal of the foreign body in the presence of only a small, smooth encapsulation and in the absence of marked emaciation.

8. Ulcer of the Stomach. Ulcus ventriculi.

Losses of tissue with undisturbed secretion of gastric juice involving the mucous membrane or deeper structures and showing a tendency to increase in size, or at least to be of long duration, are designated as true peptic or round ulcers of the stomach, whereas the nonpeptic ulcers of the stomach develop following various primary or secondary affections of the

stomach (mostly inflammation or necrosis) with disturbed secretion of gastric juice induced by destruction of tissue, and soon heal after the exciting process has been removed.

Occurrence. Ulcer of the stomach is rare in domestic animals; it is seen most frequently in the abomasum of calves (Ostertag, Kitt, Rasmussen), very rarely in the stomach of adult cattle (Kolb, Zippelius, Moussu, Gotteswinter, Kitt, Joest) also in the stomach of dogs (Johne, Fröhner, Groll), swine (Eisenmann) and horses (Münich, Jagnow, Roloff, Steinhard, Moussu, Hogard). Nagel found a round gastric ulcer in a hare.

Of 1,500 cases Tantz found ulcers in the abomasum of calves at the age of 4 weeks in 0%, from 4 to 5 weeks old in 78.4%, at 8 weeks old in 94.8%, at 10 to 12 weeks old in 95.6%, at 12 to 14 weeks old in 98%, and in yearlings in 68.7%.

Etiology. In animals peptic ulcers of the stomach always seem to result from acute ulceration or from hemorrhagic erosions. These losses of tissue may be due to any cause leading to circumscribed necrosis or insufficient nutrition of the mucous membrane of the stomach. The damaged tissue is then digested by the gastric juice and the healing of the lesions is inhibited through the continuation of the known or unknown influences. This may easily occur in deeply penetrating inflammations or hemorrhages into the mucosa, in poisoning by caustic substances, in the course of hog cholera, of chronic hog erysipelas, in tuberculosis of the stomach, malignant catarrhal fever, purpura hemorrhagica. Local disturbances in circulation with insufficient nutrition of the mucous membrane supplied by the involved vessels may be the result of thrombotic or embolic occlusions of small arteries in the wall of the stomach and in which, through lack of collateral arteries, a proper equalization of the disturbed circulation is no longer possible (artificially produced in experimental animals). Ulcers of the stomach are probably formed in a similar manner following extensive burns of the skin, and in cats following trichinosis (Lebert, Wyss). According to Fuchs, local disturbance may also result from pressure anemia, especially at the crest of the folds of the mucous membrane, particularly when the stomach is empty; according to Joest, through pressure upon the blood vessels in lymphadenosis of the submucosa (not infrequently observed in cattle and in a case of a rooster), or under certain other conditions through enlargements (tumors, chronic inflammation, infectious granulomata).

Frequently mechanical influences play a prominent part. According to Ashoff, they are also important factors in man. Into this category belong the common ulcers of the abomasum of calves occurring at the time of changing from an exclusive milk diet to a partly or exclusive vegetable diet. According to the investigations of Bongert and Tantz, this frequent occurrence is correlated with the insufficiently developed rumen in calves, as the result of which roughage, fed at the age of 3 to 4 weeks,

usually passes directly into the abomasum, which is the largest of the four stomachs at this age. The coarse particles, only slightly or not at all remasticated, produce injury to the mucous membrane, and the formation of ulcers follows. In this process a decided rôle is played by the strong constriction of the abomasum immediately anterior to the pylorus, which anatomical arrangement is specific for young calves. Because of this constriction the moving feed, meeting an obstacle, irritates the mucous membrane and causes hemorrhages and subsequent necrosis. As a result of the continued irritation, the process extends and penetrates deeper into the tissue. With advancing age and continuous development of the rumen and perfection of rumination, the irritation ceases, the extension of the ulcers is checked, and the ulcers usually heal, leaving scars which are often found in young cattle, sometimes together with ulcers. A dull force applied to the abdomen may produce gastric ulcers, because such force easily leads to hemorrhage into the gastric mucosa (Johne holds that this is the cause of gastric ulcers in calves). Injury to the gastric mucosa by gastrophilus larvæ, foreign bodies or partial rupture of the stomach wall are likewise productive of gastric ulcer.

In Stuber's experiments on dogs typical ulcers of the stomach were produced by reversing the flow of the intestinal juices into the stomach by an artificially produced insufficiency of the pylorus (digestion of the mucous membrane of the stomach by trypsin). Gudermann, Kleeblatt and others observed the formation of ulcers following the ligation of the choledochus and ductus hepatis. They attributed this partly to intoxication brought about by a disturbance in the functioning of the liver cells. (In two of the author's observations in dogs with obstruction of the choledochus by constriction, ulcers of the duodenum and stomach had formed close to the pylorus.) No observations have been recorded in animals concerning the concurrent action of hyperacidity or increased secretion of gastric juice.

Anatomical Changes. Ulcers are generally found in the neighborhood of the pylorus; they are round, oval or irregularly shaped and their base is either formed by the denuded but otherwise unchanged muscularis or serosa or by pseudomembranes covering the peritoneal coat. The margins of the ulcers, particularly those of peptic ulcers, are quite sharp as if they had been stamped out artificially. Peptic ulcers (ulcus pepticum sive rotundum, sive corrosivum, sive e digestione) do not show an inflammatory reaction in their neighborhood, they imitate in their outlines the shape of a funnel placed with its long axis obliquely towards the pylorus. If an ulcer perforates, general peritonitis occurs unless adhesions have previously been formed with the neighboring organs. Erosion of a larger vessel leads to copious hemorrhage.

Symptoms. Gastric ulcers have generally been found in domestic animals accidentally upon post-mortem examination (in slaughtering horses without preceding observation of characteristic symptoms during life). If there are any observable symptoms during life they are identical with those of chronic gastric catarrh (see page 133) or chronic atony of the proventriculi (see page 99). One may be justified in thinking of gastric ulcer if there is occasional hematemesis (vomiting of blood) or if the feces are hemorrhagic, black, tarlike. In horses mild colicky attacks have occasionally been observed. The condition of the patients often improves remarkably within a few days and then there is suddenly a marked relapse.

According to observations by Swiss veterinarians, the administration of hydrochloric acid has an injurious effect; hence this procedure, alternated with opium if necessary, might be used for diagnostic purposes (see p. 119).

If perforation occurs, peritonitis sets in and rapidly takes a *fatal course. According to Rasmussen one must think of* round ulcer if calves become restless during transportation, throw themselves down, and then lie apathetically on the floor.

Salvisberg observed sudden death in an apparently healthy and well-nourished cow (perforating ulcers). Another cow took sick suddenly, manifesting diarrhea, cessation of appetite and rumination, and grave general disturbances with fever (40° C., 104° F.), while the head and neck were held in an extended position (slaughter revealed a number of ulcers in the edematously swollen mucous membrane). In another case there was gradual progressive emaciation with uncontrollable diarrhea, while the appetite remained good (chronic ulcer). In still another case gradual emaciation was observed for weeks, followed suddenly by severe sickness, making emergency slaughter necessary. Augsburger observed the formation of fistula of the abomasum with intermittent and copious discharge of grayish green, sour-smelling contents of the stomach.

Treatment. When there is suspicion of ulcer of the stomach, undigestible, coarse feed should be avoided because it increases the danger of perforation. Mucilaginous and astringent drugs are indicated (subnitrate of bismuth, silver nitrate), also hemostatics including adrenalin or suprarenin (for dogs 30 drops of the 1% solution internally, repeated several times). The administration of hydrochloric acid is contraindicated.

Carlsbad salts in large doses or the "alkali" therapy, used to good advantage in human medicine (sodium bicarbonate in regularly repeated doses), may be used to advantage and will neutralize the acid and also inhibit the secretion of gastric juice.

Obstinate vomiting must be met by narcotics (opium).

9. Neoplasms in the Stomach. Tumores ventriculi.

Occurrence. Tumors in the wall of the stomach are very rare, and therefore, and because they are difficult to diagnosticate, they do not have a great deal of practical significance.

In the **fore-stomachs of ruminants** the omasum appears to be the favorite seat of neoplasms; here there are found polypoid tumors which attain a size of two fists and which may close up the opening between reticulum and omasum. Similar tumors are found more rarely in the reticulum. Sarcomata are likewise found in the omasum, up to the size of a fist; nodular, tough tumors, generally showing necrotic changes; these tumors are found more rarely in the reticulum or rumen. Kitt saw a chondroma in the wall of the rumen of a calf. Finally the rumen of cattle may contain ulcers as large as a hand, the base being formed by thickened nodular masses of the wall of the rumen; such ulcers are probably due to carcinomatous neoplasms. Chaussée found small tuberculous nodules in the omasum and in the reticulum of a heifer.

Tumors are common in the **stomach proper** (the abomasum), where they are found in the pylorus, the cardia, and the larger curvature. In the horse there occur sarcomata and carcinomata which, when growing in the stomach, may obstruct the cardia, the pylorus and the duodenum. In the abomasum of cattle epithelioid neoplasms are encountered, such as adenomata, adenocarcinomata and carcinomata, which undergo early degeneration; also lymphadenosis of the wall of the stomach (one case was observed in the stomach of a chicken) in the form of diffuse or circumscribed raised thickening of the wall to the extent of 2 to 3 cm. by lymphadenoid infiltration of the submucosa, whereby the wall of the stomach becomes rigid, the mucous membrane tumefied, padlike, and thrown into heavy folds, including a thickening of the plicæ spirales of the abomasum (Joest). Joest repeatedly observed in cattle tuberculous lesions originating in the submucosa and leading to the formation of extensive tuberculous ulcers. Besides these the mucous membrane of the abomasum may also show simple tuberculous ulcers originating on the surface of the mucous membrane, while the surrounding mucous membrane becomes thickened. Eberlein observed a primary carcinoma in the stomach of a dog. More rarely are lipoma, fibroma, myoma, found in the stomach; similar to the latter is the circumscribed muscular hypertrophy which is sometimes met with in the stomach of the horse.

Symptoms. The presence of a tumor of any size in the stomach disturbs its function on account of the destruction of part of the mucosa and on account of the impediment to the proper movements of the organ. Disturbances of digestion are, therefore, observed, which, however, have nothing characteristic, but are identical with those seen in chronic gastric catarrh or in atony of the fore-stomachs. In horses symptoms of colic are also seen occasionally after the ingestion of food; further, dizziness and vomiting; in cattle chronic bloating, frequent belching, vomiting; in dogs enlargement of the circumference of the

abdomen, vomiting, icterus. The animals become more and more emaciated, cachexia sets in and they finally die after complete exhaustion. Exceptionally a disintegrating neoplasm may lead to perforation of the stomach wall or to internal hemorrhage (Menges) in consequence of the erosion of a larger blood vessel.

Diagnosis. In large animals, gastric tumors can hardly ever be diagnosed with certainty. Exceptionally a tumor may be supposed to exist in horses, if palpation through the rectum discloses a firm thickening of the blind sac of the stomach, and if obstinate digestive disturbances are present; in some cases the extension of the tumor to neighboring organs and especially persistent, severe icterus are present. In carnivora, tumors are rare; they are located preferably in the regions of pylorus or fundus, and may be made out by palpation; this shows a firm, uneven body, not sensitive to pressure, which is slightly movable and lies behind the ensiform cartilage in the median line, or to one side. Large tumors move synchronously with the respiratory movements, being in contact with the posterior surface of the liver; they may be traced out to the frontal plane of the last rib. The symptoms are emaciation, vomiting after eating, splashing sounds in the stomach on thrust-palpation, occasionally severe icterus. If the tumor breaks down, the microscopical examination of vomited material or of lavage, as well as the chemical analysis may be of value.

Treatment. Only in carnivora is there any hope of recovery if the operative removal of the tumor can be carried out successfully. Parascandolo performed gastrectomy in a dog with good success.

10. Torsion of the Stomach. Torsio ventriculi.

(*Volvulus ventriculi.*)

In dogs the dislocation is such that the pylorus passes along the ventral abdominal wall around the end of the esophagus from right to left; the large curvature and the fundus pass along the dorsal abdominal wall to the right and forward, whereupon the pylorus comes to lie in the left hypochondrial region between the liver and cardia, while the fundus of the stomach is compressed. The end of the esophagus is closed by torsion around its long axis, the spleen is attached to the large curvature of the stomach, and these organs are placed crosswise and horizontal in the abdominal cavity with the spleen bent like a horseshoe, its dorsal end and the fundus of the stomach being located in the right hypochondrial region. This dislocation, besides causing obstruction of both openings of the stomach, rapidly brings on, by compression of blood vessels, an intense venous hyperemia with serohemorrhagic infiltration of the walls of the stomach and a strong distention of the

stomach. From past observations torsion of the stomach occurs not infrequently in dogs and almost exclusively in older dogs of the larger breeds. Dogs used for draft purposes seem to suffer most frequently from the affliction, a circumstance which seems to explain the fact that this condition occurs less frequently in Hungary, where dogs are not used for this purpose than in other countries. Only one case of torsion of the rennet in a calf eight days old has been reported by Carougeau and Prestat.

Etiology. The frequent occurrence of torsion of the stomach in dogs is due to the greater mobility of the pyloric end. According to observations and experiments made by Poenaru and Slavu and Marcus, this torsion is greatly increased by the relaxation or even rupture of the ligaments of the stomach, especially of the gastrohepatic and the gastroduodenal ligament, which follows repeated overloading of the stomach in older dogs. Torsion is most frequently caused by turning and rolling movements, or movements combined with extreme lateral displacements of the body, particularly rolling, rapid running down stairways and jumping. In these movements the pyloric end of the stomach, attached to the esophagus as a fixed axis, is actuated into oscillating, swinging dislocation within the abdominal cavity like the ball of a pendulum by movements of the point of attachment of the string of the pendulum. This is likely to occur if the stomach is only partially filled, mostly with liquid contents or especially with hard and heavy material (pieces of bone, meat and liver, potatoes), and is surrounded by only partly filled or empty intestines, and thus is not prevented from executing such dislocations. When the stomach is well filled such dislocations can not occur because it is held in place by the pressure of the abdominal organs and the abdominal wall. On the other hand, when the stomach is empty the inertia of the small, light organ is too slight for it to be brought into swinging motions.

In ruminants torsion of the abomasum may, according to Magnusson, be explained by the anatomical arrangement, since in young animals the abomasum, running with the pyloric end toward the opening between the abomasum and the omasum, is really held in position solely by the great omentum. When the abomasum is filled any violent movement (jumping, rolling) may rupture it, and it then becomes twisted around its suspending apparatus.

Pathogenesis. In consequence of change of position, both openings of the stomach become closed; the veins of the stomach and those of the spleen, which is forced to follow the change of position of the stomach, become partially or completely obstructed in consequence of pressure and torsion. The less collapsible and more resistant arteries, in which there is a high blood pressure, continue to propel an almost normal amount of blood into the vessels of the stomach and spleen,

and a rapidly increasing profound venous congestion is developed. Excess of carbon-dioxide in the gastric vessels causes convulsive colicky pains and contractions of the gastric wall, which will only cease after several hours and after bloating and edematous infiltration of the gastric wall have developed. Pain is also caused by a torsion of the nerves and by the development of peritonitis. Since nothing can pass from the stomach and since absorption into the blood is almost abolished, the stomach becomes quickly and enormously dilated by gases which develop from food stuffs that may be present, and from the hemorrhagic infarction with serum extravasation, which has been caused by the torsion. The more feed there is present in the stomach, and the more severe the twisting, the more rapidly will bloating attain a dangerous degree. The enlarging stomach interferes with the movements of the diaphragm and in this manner and also in consequence of a developing general infection, eventually also of peritonitis, which is caused by microbic invasion through the damaged stomach wall, disturbances of circulation are produced.

Anatomical Changes. The pyloric half of the sometimes tenfold balloon-like disteuded stomach, including the first portion of the duodenum, is found on the left between the posterior surface of the liver and the now anteriorly directed posterior surface of the stomach below the slightly spirally twisted esophagus, the large curvature of the stomach being located in the right hypochondrial region. The mesentery of the duodenum forms a tense string drawn between the right kidney and the stomach and compressing the median part of the latter from in front. The mesentery is drawn over the dilated stomach and the dark red wall of the latter may be seen shining through it. Upon cutting open the stomach, gas is discharged abundantly; the mucosa is dark red; the whole wall is infiltrated and edematous. Not infrequently one can observe that the muscle bundles are beginning to part (Joest); very seldom a rupture is found (Ellermann). The splen is greatly enlarged, bent in the shape of a horseshoe, often sharply kinked in the middle, blackish red in color, its capsule tense. The abdominal cavity usually contains a bloody, serous fluid and the liver is somewhat displaced to the left and anemic. Incipient peritonitis is found ocasionally and lesions of asphyxiation are always found.

In a case observed by Magnusson in a lamb a total torsion of the abomasum to the right was found. The point of torsion was situated, on the one hand, at the constricted part between the omasum and the abomasum, and on the other hand about 15 cm. behind the pylorus. The great omentum was ruptured.

In the case reported by Carougeau and Prestat, the abomasum of the calf was twisted towards the right; both open-

ings were closed; its wall showed evidences of much congestion the peritoneum was inflamed.

Symptoms. The disease is characterized by a sudden attack of abdominal pain, and by rapidly increasing bloating. The patient stands motionless and apathetic on one spot and if urged on, moves stiffly and carefully. The ingestion of food and drink, and defecation cease; exceptionally there may be intense thirst (Bonvicini), but the ingested water at once returns through the mouth or nose. Gagging sometimes supervenes but vomiting does not occur; irritation of the fauces causes retching, but no vomiting. Jakob succeeded in one case, in spite of a torsion of 360°, in passing a rubber tube into the stomach. (There was only light strangulation, and for that reason also only light signs of engorging of the blood vessels after death.)

Another striking phenomenon is the increasing distention of the abdomen in all directions, although most markedly in the middle mesogastric region, while the percussion sound is everywhere loud or highly tympanitic (occasionally of a metallic character), with the exception of a dullness about the size of the palm of the hand in the right hypochondric region, caused by the spleen resting against and slightly bulging the abdominal wall at this point (Cadéac, own observations). The distention usually increases rapidly, but in exceptional cases (Bonvicini) it may increase slowly. The abdomen is sensitive to pressure and in the abdominal cavity a tense sac may be felt. During jerky shaking of the lower gastric region or during rapid change of position of the dog, splashing can be plainly heard (Jakob).

As the abdomen gradually increases in size, difficulties in respiration increase to attacks of suffocation; cyanosis becomes marked, the heart beat is much accelerated, hard; the pulse becomes gradually weaker and finally it can no longer be felt.

The disease is of short duration, death ensues within 24 to 48 hours after the appearance of the first symptoms, as a rule in consequence of suffocation. In cases of light strangulation the animal may, in spite of a torsion of 360°, live for five days (Jakob).

After successful reposition of the dislocated gut a recurrence is possible even after five months (Larsen).

In the case of Carougeau & Prestat, the previously healthy calf stopped sucking, protruded its tongue, did not lie down, defecated less frequently, colicky pains and constipation soon came on. After the administration of milk the restlessness increased. The next day weakness was already well marked and pressure upon the abdomen elicited splashing sounds. After a day and a half the calf died. In the case of Magnusson the lamb died suddenly.

Diagnosis. The clinical picture in the dog is characteristic enough to enable us to make a diagnosis; an exploratory punc

ture may however become advisable. In obstruction of the esophagus there are no abdominal pains nor bloating. In displacements of the intestines there occurs true vomiting, bloating of the abdomen is absent or quite insignificant. In the course of general acute peritonitis vomiting is frequent, fluids poured into the mouth get into the stomach, bloating is not very marked and there is fever from the start.

Treatment. According to Jakob, success does not follow the tapping of the stomach a hand's width below the highest point of the distention to evacuate the gases slowly, nor the passing of a stomach tube in case of incomplete torsion of the esophagus to permit the escape of gases, followed by the introduction of 1 to 2 liters of fluid and the forceful rolling of the dog from right to left to bring about a reposition of the torsion, even with simultaneous assisting massage of the abdominal wall. On the other hand, an operation may lead to a cure in less advanced cases. To reduce the tension of the abdomen and to relieve the lungs, the gases should first be evacuated from the stomach by tapping, after which a long incision is made along the median line and the fingers are introduced on the left side between the stomach and the liver and the pylorus and duodenum drawn back to their normal position. The evacuation of a bloody, serous fluid with the gas on tapping is a very unfavorable sign. In one case, which, however, has not been thoroughly explained, Cadéac achieved a cure following laparotomy. In a case of Jakob all attempts at reposition were fruitless because the spleen, contrary to the general rule, was displaced in front of the stomach and the ventral left lobe of the liver and did not follow the artificial turning of the stomach. In Larsen's case, however, the rectification of the displaced organs was easily accomplished, and this was also true following the recurrence of the same affliction in the same dog 7 months later.

Other Changes of Position of the Stomach. In dogs, the stomach sometimes slips through an opening in the diaphragm into the thoracic cavity (Fünfstück, Johne, authors' observation); a case of Baerner shows that the horse's stomach may also exceptionally get into the thoracic cavity. In ruminants, the reticulum and occasionally the omasum may enter the thoracic cavity through an opening of the diaphragm. A prolapse of the stomach in a hog, which appeared to be healthy during life, was found by Fritz.

An auto-invagination of the stomach was reported by Adam, invagination of the stomach into the duodenum by Spencer.

If strangulation of the prolapsed stomach takes place, the animals which have already suffered from difficult respiration succumb in a short time to suffocation. In the case of Baerner a roaring horse with a partially prolapsed non-incarcerated stomach had occasional attacks of colic. In ruminants, prolapse of the fore-stomachs causes chronic bloating, possibly also compression of the heart.

11. Hemorrhage from the Stomach. Haemorrhagia ventriculi.

(*Blutbrechen*, [German]; *Hæmatemesis.*)

Etiology. Arterial hyperemia of the gastric mucosa easily causes hemorrhages per diapedesin or per rhexin, which are rarely so abundant that there is a free blood extravasation into the cavity of the stomach. This may however be the case if acrid or caustic substances have caused a deeply penetrating inflammation or a necrosis of superficial layers of the mucosa with subsequent ulceration.

Hemorrhages from the stomach likewise appear in the course of certain acute infectious diseases; they may then be due to local inflammatory processes or more generally to the general infection. Diseases of this type are particularly purpura hæmorrhagica, anthrax, hog cholera, swine plague, distemper in dogs, smallpox, scorbutus, etc.

Diseases of the blood vessels and changes in the blood are the cause of hematemesis which occurs in blood diseases and cachexia. Ulcer of the stomach gives rise to hemorrhages from the stomach. Swallowed foreign bodies sometimes injure vessels of the gastric wall and cause hemorrhages into the stomach, gastric vessels may also be torn in a fall or in overwork. In horses gastrophilus larvæ sometimes produce injuries which lead to hematemesis (Qualitz). Rupture of the enlarged and subsequently adherent abdominal aorta or of the cœliac artery may also form an extremely rare cause of fatal hemorrhage into the cavity of the stomach. In Neven's case a horse bled to death into the stomach because, through simultaneous necrosis of the wall of the carotid artery and of the esophagus following gas gangrene, a perforated adhesion was formed between the carotid artery and the esophagus. Congestion of the gastric mucosa leads usually at most to parenchymatous hemorrhages, as is seen in the course of diseases of the liver, of the portal vein, of the heart and of the lungs.

Symptoms. The only reliable symptom of hemorrhage of the stomach consists in the vomiting of blood (hæmatemesis, sive vomitus cruentus); it is however usually observed only in carnivora and then only after more severe hemorrhages. The animals then expel either feed-material streaked with blood or pure blood through the mouth and nose. The latter may be fluid and bright red or coagulated into dark brown masses. After having been in the stomach for a longer time, the vomited blood is dirty brown or of a chocolate color, similar to ground coffee, since the hemoglobin has been changed into hematin by the action of the hydrochloric acid of the gastric juice. Microscopic examination of the vomited masses which have an acid reaction or of the gastric contents removed by catheter shows,

aside from more or less changed particles of food, pale or disintegrating blood corpuscles, although they may be entirely absent, having been completely dissolved by the gastric juice.

Most frequently blood which has been extravasated into the cavity of the stomach enters the intestinal tract and leaves the body with the feces. During its way out it is always changed materially; the hemoglobin is decomposed and the albumen undergoes putrefaction, hence the feces are tar-like discolored, soft, or on the contrary dry and very fetid.

If the hemorrhage is profound the general symptoms of internal hemorrhage become manifest; pallor of the mucous membranes, small pulse, perspiration, dizziness; repeated small hemorrhages lead to chronic anemia.

Diagnosis. Gastric hemorrhage can be diagnosticated only upon the basis of hematemesis, or upon the removal of blood-tinged masses with the stomach tube and then only if it can be ascertained that the blood comes from the vessels of the stomach and did not get into the latter from some other place. The difference between pulmonary hemorrhage and hematemesis has been pointed out elsewhere. It should here again be mentioned that blood may get into the stomach from the nose, from the buccal cavity and from the respiratory tract; such hemorrhages must be excluded before a diagnosis of gastric hemorrhage can be made reliably. There is also the possibility that dogs may vomit previously ingested blood; this is often indicated by the fact that no anemia can be observed in spite of large masses of blood vomited. Whether blood voided with the feces comes from the stomach or from the anterior portion of the intestinal tract cannot be determined solely by the appearance of the feces; the clinical history of the case and the examination of all other organs must determine the diagnosis.

Treatment. In order to stop the hemorrhage the patients should be placed at rest and should be made to swallow ice or some cold water; in dogs external applications of ice may be made in the region of the stomach. Subcutaneous injections of ergotin (5-10.0 gm. or 0.2-1.0 for small animals), extr. hydrastis (8-10.0 gm. or 0.2-0.5 gm.) are used with advantage. To diminish the movements of the stomach opium internally (5-10.0 or 0.1-0.5 gm.) is useful, eventually combined with astringents (acetate of lead, tannin, tannoform, tannalbin, sulphate of iron, chloride of iron, alum). Gelatin may also be used either alone or with wine internally; also tonogan, suprarenin, paranephrin (for horses 0.02 to 0.04 cc. of 0.1 solution per kg. body weight, or, on the average, a total dose of 5 to 15 cc. subcutaneously, 0.01 cc. per kg. body weight, or a total dose of 1 to 5 cc. intravenously; for dogs a total dose of 0.2 to 0.5 cc. sub-

cutaneously). Caution is necessary in internal medication since it may further stimulate vomiting.

As long as the hemorrhage lasts, the animals should be starved, then they should receive only fluid, soft and mashy feed for several days.

12. Intestinal Hemorrhage. Enterorrhagia.

Etiology. More severe hemorrhages upon an inflammatory basis are usually observed only if the inflammation has been caused by an infection or by acrid caustic materials or if it is complicated by ulceration. This is seen in the course of anthrax, cattle plague, hog-cholera, swine plague, distemper in *dogs, pyemia, septicemia; after intoxication by caustic poisons,* in meat poisoning, after the administration of drastic laxatives. Intestinal hemorrhages are frequently seen during purpura hemorrhagica, scorbutus, and other diseases, and are due more to morbid changes of the vessel walls than to a local disease of the mucosa.

Mechanical injury of the intestinal mucosa by swallowed foreign bodies (fragments of bone, nails, etc.), animal parasites, echinorrhynchus gigas, taenia echinococcus, dochmius duodenalis, etc., desiccated coproliths, hard substances introduced into the rectum (end-piece of a rectal syringe), or stabs produced intentionally (Sequens) may cause greater or lesser intestinal hemorrhages.

Congestion of the intestinal veins in chronic interstitial hepatitis or in valvular disease of the heart, obstruction or compression of the portal vein, thrombosis or embolism of intestinal arteries may occasionally be the cause of intestinal hemorrhages. Hemorrhoids (dilatation of veins) below the rectal mucosa, which is sometimes seen in dogs and horses (Utz, Eckart) also are the cause of hemorrhages from the rectum, and for the development of which lack of exercise, drastic purgatives, constipation, irritating food, difficult defecation and proctitis constitute favorable conditions, especially in old, fat animals (Schreck).

As further causes of intestinal hemorrhages there may be mentioned: very vascular or disintegrating tumors, especially mucus polyps, intussusception, tearing of the dilated abdominal aorta or of the anterior mesenteric artery after they had previously become adherent to the intestinal wall, finally extensive burns, probably on account of the formation of emboli in the intestinal vessels.

Symptoms. The feces are stained with blood, or pure blood may occasionally be voided through the anus. The more profuse the hemorrhage, the more easily blood-coagula are formed, so that the voided masses may sometimes consist of coagula only. This is particnlarly observed in hemorrhage from the posterior portions of the intestines, since blood here does not

mix well with the already firm feces. Hemorrhoids cause repeated hemorrhage in the rectum and itching in the anal region; bluish red knots up to the size of a nut are encountered on the outside of the anus (external hemorrhoids) or on the inside of the anus (internal hemorrhoids) (Schreck). In hemorrhage in the anterior portions of the intestines, the feces are intimately mixed with blood, they are tar-like, black, or even as though charred, and they have an intense putrefactive smell. If the hemorrhages have been only slight the feces appear pale reddish or only streaked with blood; in slight rectal hemorrhages the bloody streaks are limited to the surface. Defecation may be very easy or it may be quite difficult, particularly if the hemorrhage is due to inflammatory conditions.

It is possible to demonstrate the presence of red blood corpuscles or of hemoglobin, even in occult bleeding, by microscopical or chemical examination, although the appearance of the feces may be normal. If the bleeding is more than slight the addition of water to the feces will afford information, because the water is colored red after the fecal matter has sedimented.

For a microscopical examination, lentil- to pea-sized particles of the fecal matter are taken from several places, or several separate large drops if the feces are thin and mushy; they are stirred together with some physiological salt solution and spread thin on a slide. With a sufficient magnification, the red cells can be seen either uninjured or swollen and pale, more frequently fallen apart; they are, however, missed not infrequently, especially in hemorrhage from the anterior portions of the intestine, because they have been destroyed during their passage.

The chemical demonstration of hemoglobin is usually positive, even in those cases of gastric or intestinal hemorrhage where the microscopical examination fails, because the coloring matter of the blood can be shown to be present, by chemical means, even in a dilution of 1:150.000-200.000. Since the blood test may be positive as long as four days after feeding meat or dog-biscuit, carnivora must be fed with vegetable food and milk for 4-5 days before the examination. The following tests are adapted to the demonstration of hemoglobin:

The *Benzidin-test* of Schlesinger & Holst, in which a pea-sized portion of the feces is placed in a test tube filled one-fifth with water, and brought to a boil, in order to destroy oxidizing ferments which may be present accidentally and may simulate a positive reaction. Ten to 12 drops of a freshly prepared solution of Benzidinum purissimum, Merck, are mixed with 2½-3 c. c. of a 3%-solution of peroxide of hydrogen (of the benzidine take the point of a knife full to 2 c. c. of glacial acetic acid); to this mixture about 5 drops of the cooled suspension of fecal matter are added. Usually the mixture immediately assumes a beautiful green or blue color, or if very little blood is present, after ¼-½ minute, and on standing it becomes deep violet.

For the *Guaiac-test*, a fine paste is made by rubbing up the fecal matter carefully with water; one-third of the volume of glacial acetic acid is added to the paste and the whole is extracted with ether; 3-5 c. c. of the extract are placed in a test tube, with 10 drops of a freshly prepared tincture of guaiac and 20 drops of old oil of turpentine. On shaking, the color turns blue-violet or greenish blue. According to Wetzel, this test is negative after eating meat, and for this reason a positive result indicates a pathological process.

The *aloin-test* also is made with an ether extract of the fecal matter, to which are added 20-30 drops of turpentine and then 10-15 drops of an alcoholic solution of aloin (a small knife-point full of aloin in 3-5 c. c. of 60-70% alcohol). The appearance of a light-red and later cherry-red color proves the presence of blood.

In the *paraphenylendiamin-test* after Boas, the ether extract of the fecal matter is mixed with 1-2 drops of a ½% solution of paraphenylendiamin; an olive-green color appears almost immediately on shaking, and turns promptly dark violet. According to Wetzel, this test also is negative after eating meat.

Diagnosis. As a rule blood can be recognized in the feces with the naked eye. One may however be misled by a naturally dark brown color of the feces of carnivora after eating meat; or by the brown color of feces of animals which have received preparations of bismuth or iron. In these cases the water which is mixed with the feces is not colored red, and the microscopical and chemical examinations are negative. In order to avoid these sources of error, all meat or dog-biscuit should be withheld for at least four days before a chemical examination is to be made, and the animals must not be treated with bismuth or iron during this time, the pale-red color of the feces of plant-eating animals, after feeding with red beets, disappears on the addition of potassium hydrate to a suspension of the fecal matter.—Hemoglobin may be demonstrated microscopically, or only by chemical examination, in the feces of animals which have eaten of the blood of slaughtered animals or have licked bleeding wound; but in these cases there are no signs of internal disease.—Hemorrhage from the stomach causes the same symptoms as that from the intestine, and if the blood is intimately mixed with the feces or if the later have a uniform tar-black color, only an exact examination of all organs, together with a careful consideration of the clinical history can decide the question.

The site of the bleeding is indicated to a certain extent by the color of the discharged blood, in so far as the blood is altered the more, the closer to the stomach the bleeding has taken place. The presence of streaks of blood on the surface of feces, which otherwise are normal, indicates rectal bleeding; close admixture of blood and feces suggest bleeding in the upper portion of the colon or in the small bowel. It is to be kept in mind, however, that the macroscopical and even the microscopical demonstration of blood in the feces may fail in spite of an existing intestinal hemorrhage, either because the trouble has lasted only a short time and the discharged blood has not reached the anus, or because a mechanical obstruction renders the removal of the blood toward the anus impossible, as is the case for instance in the course of intestinal torsion and strangulation, not infrequently in intestinal invagination and in severe cases of thrombosis of the mesenteric artery.

Treatment. The treatment of intestinal hemorrhage is in general identical with that of hemorrhage from the stomach, and preparations of opium and astringents may be administered every one to two hours without any fear. Hemorrhages from the posterior portion of the intestinal tract require local treatment from the rectum with gelatin dissolved in hot water, astringents, also adrenalin or suprarenin. If the loss of blood has been considerable, intravenous injections of physiologic salt solution may be very beneficial.

Rectal Hemorrhage of Cattle. (Hemorrhagic inflammation of the rectum, "Rücken-" or "Lendenblut," [German].) Haubner has described a hemorrhage from the rectum in ruminants, particularly in cattle, which is sporadic or enzootic in character; its anatomical cause is claimed to be a moderate inflammation of the rectum brought about in well-fed animals by the ingestion of irritating acrid vegetable feed.

Holterbach has recently reported several cases of this affection in young cattle of poor owners, also in larger herds. He observed at the same time numerous cases of rectal hemorrhage in dogs.

The **symptoms** are diminished appetite, depression, stiffness of the back, and tenesmus. After one to two days, dry firm feces, mixed with blood, are voided with an effort; sometimes pure blood. The rectum is hot and swollen. In spite of an elevation of temperature (up to 40.2° C.) there is no change of pulse or respiration. The disease usually ends in recovery within two to five days; it may, however, exceptionally lead to a fatal termination.

The **treatment** recommended by Haubner, consists in the administration of salts, and in careful irrigation and emptying of the rectum with mucilaginous fluids, or dilute vinegar and cold applications to the region of the sacrum. Holterbach gives, every two hours, a tablespoonful of the following mixture: Ferri sulphat. sicc. pulv. 150. gm., Sal. Carolin fact. pulv. 300. gm., Pepsin Witte. Germ. 30. gm.

13. Acute Intestinal Catarrh. Catarrhus intestinalis acutus.

(*Katarrhalische Darmentzündung* [German]; *Enteritis catarrhalis acuta.*)

Under the term intestinal catarrh are included not only the inflammatory changes in the intestinal mucosa causing a disturbance of health, but also diarrheal discharges due to a purely functional disturbance of the movements and secretions of the intestines, since a hard and fast line can not be drawn clinically between the two, and transitional forms also exist.

Etiology. The etiology of acute intestinal catarrh is on the whole identical with that of acute gastric catarrh (see page 124). Dahne saw several cases of acute intestinal catarrh with grave general symptoms after the ingestion of Swedish clover which had grown abundantly and luxuriantly on a pasture. Cattle often show signs of acute intestinal catarrh with profuse diarrhea after the ingestion of green leaves of sugar beets. Poisoning of cattle by turnips, which has frequently been observed in Denmark (see page 97), also manifests itself partly by an acute intestinal catarrh. A similar observation was made by Sustmann in horses that had consumed daily rations of 6 to 20 lbs. of beet root (Runkelrueben) and sugar beets, when a bloody intestinal catarrh followed. Menneking and others repeatedly observed an acute catarrh and dilatation of the stomach in horses after feeding with soaked chopped sugar beets. The molasses disease frequently observed by Schlegel in heavy draft horses also manifested itself partly in an acute catarrh of the stomach, in which the disturbance did not appear

until 3 weeks after feeding began (daily 1,250 grams and more of molasses). According to Ellenberger & Waentig, the often observed gastrointestinal catarrh and colic following the feeding of molasses and cane sugar (mixed with cut fodder as so-called "Futterzucker") are due to feeding these materials in too large quantities or in improper proportions. Some horses, however, are very sensitive in this respect, even when only small quantities are fed. The disturbance was probably due partly to the noxious effect of feeding spoiled or fermenting feed. In sporadic cases, acute intestinal catarrh could be traced to feeding bread without the addition of sufficient quantities of other feed (Grundmann). Nevermann and Lorscheid observed a severe intestinal catarrh and enteritis in horses on the next and second day following the purchase and feeding of a new batch of bran in a daily ration of 3½ kg. In the German army horses, cases of acute gastrointestinal catarrh with colic were frequently observed and attributed to the feeding of a mixture of wheat and oats containing admixtures of dirt and other material. Naumann saw many cases of sickness accompanied by founders in horses fed on rye (horses fed with freshly threshed rye became much sicker than those fed rye in sheaves). A bloody and often fatal intestinal catarrh was repeatedly observed in horses fed wheat that had been treated with copper sulphate for the prevention of smut. Similar observations were made by Eber in pigeons, which under certain circumstances often died in large numbers from a bloody gastrointestinal catarrh after feeding on treated seed in the field, especially in fall and spring (the substances used in treating the seed for the prevention of smut often contain arsenic).

Microorganisms frequently play an important part in the production of the affection, particularly saprophytes, which are present normally, if their virulence is increased or the resistance of the intestinal epithelia is diminished. Intestinal catarrh is produced in this manner after the ingestion of rotten or sour feed, as for instance sour beer draffs, after the ingestion of meat from sick animals, which is infected with pathogenic bacteria. Pflüger and Uebele claims that dogs sometimes become sick with acute intestinal catarrh if they receive exclusively horse meat which is otherwise unobjectionable. These attacks may even run a fatal course, and it is claimed that the toxic element can be demonstrated in the bouillon from such meat and that it may be made innocuous by sheep or cattle fat.

In this case, the question of anaphylaxis may arise, due to enteral introduction of foreign proteins, as Rosenau & Anderson succeeded in producing it artificially in guinea-pigs by the intestinal introduction of horse-meat or horse-serum. (Perhaps this affords an explanation of the frequent occurrence of serum-disease among the tartar population of Russia where horse-meat is a principal article of diet.)

In serum sickness a catarrhal, hemorrhagic or even pseudomembranous enteritis is a frequent occurrence (Widmer). A similar condition may be produced by intravenous injections of bacterial suspensions (colon bacteria, typhoid bacilli) in which the cytolysis of tissue of the organs probably furnishes the material for the production of poisonous substances of catabolism. (Similar conditions might also occur occasionally under natural conditions after absorption of bacteria from the intestinal tract.) An independent infection is probably responsible for intestinal catarrh which occurs in larger establishments, especially in herds of hogs, independently of dietary errors, and which may affect many animals within a short space of time. (Aronsohn, Esser, the authors.)

An infection must be assumed to be the cause of the affection if in the absence of errors of diet, numerous animals of a large herd sicken within a short period of time, as occurs frequently among herds of swine (Aronsohn, Esser, authors' observation). In young fowls we observe an enzootic intestinal catarrh which is probably due to infection (Klee).

Very fibrous vegetable parts which are not, or only with difficulty, digested, as well as ingested sand, sometimes injure the mucosa, while the great majority of laxatives will only produce a catarrhal condition in the intestinal mucosa.

External and internal cooling of the body does not only cause energetic peristalsis by reflex irritation, but it may also produce an intestinal catarrh; this is evidently the cause of the frequency of this affection during the colder season (pasturing on frosted meadows, feeding on frozen potatoes, beets, the ingestion of ice-cold water), while the not uncommon occurrence of the disease during the warmer season (summer diarrhea) is probably usually due to the ingestion of impure water. Working oxen sometimes develop short attacks of intestinal catarrh during the summer if they drink too much water while heated (Cruzel).

As further causes must be mentioned, intestinal worms, intestinal calculi, desiccated feces, the irritating effect of which may be further increased by the presence of bones and other foreign bodies. Catarrh of the rectum may be due to improper manipulation in examination and treatment.

Intestinal catarrh is often observed to be **secondary** to acute infectious diseases; it also presents itself almost constantly as one of the symptoms of septicemia.

Pathogenesis. Many of the above noted conditions, either directly or by the formation of irritating products of fermentation or putrefaction, cause only an increased peristalsis and often an increased secretion of fluid into the intestinal canal, with diarrhea, without setting up an inflammatory process in the intestinal mucosa, if, as a result of the diarrhea, the in-

jurious substances are in time removed from the intestinal tract, or, as occurs in at least some cases, transported from the small intestines to the less sensitive large intestines.

In most cases the intestinal mucosa is soon injured by mechanical, thermic or chemical influences. The inflammation sets in with degeneration of the epithelium followed by dilatation of the capillaries in the intestinal mucosa and simultaneously the excretion of an albuminous fluid into the lumen of the intestines. This is accompanied by increased intestinal secretion and by the migration of leucocytes into the tissue and into the lumen of the intestines.

The intestinal function is affected by the catarrh in various directions. Above all a marked, abnormal disintegration of the intestinal contents occurs, especially an acid fermentation with the formation of lactic, butyric and acetic acids, also the development of gas (carbonic acid, hydrogen, methane), putrefaction of proteins, associated with the formation of sulphuretted hydrogen, indol, skatol, etc. The cause for these processes is found in the fact that the constituents of the chyme are not accessible to the digestive enzymes, being insufficiently prepared mechanically and chemically, further in the great multiplication of the intestinal microorganisms, in the insufficient formation of digestive enzymes, in the secretion of an albuminous fluid which is subject to disintegration, and in the fact that the process of absorption is disturbed owing to the injury suffered by the intestinal epithelia, and partly on account of the increased peristalsis. The resulting products of disintegration contribute their part in aggravating the inflammation. The irritating action of most of these products, upon the inflamed intestinal mucous membrane, which is more sensitive for the same reason, accelerates the intestinal movements, which at times may become convulsive and may thereby lead to colicky pain. The increased peristalsis, the disturbances in the absorption and the exudate which is poured into the lumen of the intestine give rise to diarrhea, if the catarrhal inflammation has involved the colon of herbivora, and at least the distal portions of the small bowel in carnivora.

The general effects of intestinal catarrh are manifested particularly in weakening of the body because of the disturbances in digestion and assimilation, and also because of the exudate into the interior of the intestine, the amount of which corresponds with the extent of the inflamed mucous membrane. Young animals with rapid metabolism, emaciated animals which are feeble or old and do not possess a sufficient supply of stored-up energy, are debilitated rapidly, particularly if the small intestine is involved and, in herbivora, the cecum. The loss of strength is due in part also to the loss of water which is considerable in profuse diarrhea and causes the blood to become thickened, disturbs the processes of metabolism in the tissue cells,

impedes the circulation of the blood by increasing its viscosity. Further, the intestinal catarrh may give rise to the absorption of toxic substances, if the intestinal epithelia and the digestive enzymes are markedly injured in their function of destroying protein toxins which may have been ingested with spoiled food or may have been formed in the course of the abnormal disintegration in the intestine. In cases of this sort, considerable quantities of bacterial endotoxins sometimes are absorbed and produce fever. The bacteria themselves can also be absorbed if the intestinal epithelia are greatly injured; in consequence the microorganisms are either dissolved forthwith, or they are deposited in accidentally predisposed organs and, in the latter case, produce local inflammations of distant organs. In case of severe degeneration or partial desquamation of the intestinal epithelial cells, foreign proteins may finally be absorbed and thereby lead to anaphylaxis, more frequently in the form of urticaria, which, however, may also be due to an injury of the blood vessels by amines originating from decomposition of protein in the food.

Anatomical Changes. Sometimes throughout its whole extent, sometimes in some parts only, the intestinal mucosa is reddened, swollen and abundantly covered with mucus; it sometimes also shows small hemorrhages. The submucosa is in a condition of edematous infiltration. The villi are likewise swollen; they are crowded together and give a velvety appearance to the intestinal surface. The solitary and agminated follicles are swollen and sometimes discharge a purulent mass on pressure, or their surface has become changed into a round ulcer. Especially in the large intestine the mucosa shows a fine bran-like deposit indicating necrosis of the epithelial cells. The feces are fluid or thin; even in the small intestines they are mashy, very fetid and contain threads of mucus, pus, blood. The mesenteric glands are in a condition of acute swelling.

Symptoms. If there is also catarrh of the stomach the symptoms of the latter predominate, especially lack of appetite and listlessness, and particularly at the beginning. The most prominent symptoms of acute intestinal catarrh consist in changes in defecation and in the character of the feces. In herbivora, constipation or retarded defecation is often observed for days and occurs in consequence of the preceding catarrh of the stomach. In a few cases, and always in carnivora, the intestinal catarrh commences with diarrhea; the latter is rarely absent in carnivora, more often in herbivora, because in them the affection may be confined to the small intestine. The feces are mushy or entirely fluid; they contain poorly digested foodstuffs, also frequently mucus and shreds of the epithelial lining, sometimes streaks of blood. They are very fetid, and usually have an acid reaction. Feces of this nature contaminate the

region around the anus or of the cloaca in fowls; they are dropped frequently and in large amounts with signs of tenesmus; later involuntary passages may take place. When the animals defecate the mucosa of the rectum sometimes prolapses and later there is a relaxation of the sphincter muscles with gaping of the anal opening.

The abdominal circumference either remains normal or is diminished; in other cases it becomes larger. Palpation sometimes elicits pain, especially in small animals. The intestinal sounds are more lively; they are often audible continually, very loud, rumbling, so that they may be heard for a distance of several feet; they are markedly metallic. Fetid flatus sometimes escapes from the anus with loud sounds.

Convulsive contractions in the intestines of the horse, occurring at short intervals, cause colicky pains which increase after the ingestion of food or drink, and they manifest themselves differently in various animals. Horses may show milder or more severe colicky pains. Cattle manifest pain by dull roaring, groaning, kicking with their hind legs towards the abdomen; dogs hide themselves, lie curled up, howl loudly from time to time, jump up suddenly and run backward and forward; hogs hide in the straw, grunt loudly and frequently roll about; fowls do not show any manifestations of abdominal pain.

The urine is diminished, dark in color, and in carnivora furnishes a rich deposit of urates; its reaction is acid even in herbivora; indican is increased.

The temperature is frequently somewhat elevated. In more serious cases and in severe diarrhea the heat is unequally distributed at the periphery; the pulse is accelerated. *Thirst is usually increased.*

If the intestinal catarrh is severe and if diarrhea continues for any length of time, prostration sets in rapidly. The animals now lie motionless on the floor or they are hardly able to stand on their feet; the eyes sink in, the glance becomes lusterless, the hair rough; fowls drop their wings, stand or sit long in the same place; the exhaled air is bad and the whole body has a similar fetid smell. The abdomen is drawn in and tender to pressure; the temperature and the pulse become subnormal, the heart beat becomes hammering, and death now soon ensues.

If the **small intestines** alone are affected (Ileitis et jejunitis catarrhalis acuta) diarrhea may be absent and the feces may appear fairly normal; the intestinal sounds are more lively than under normal conditions, especially in the region of the small intestines; violent pains may occur frequently. Disease of the duodenum may be assumed if there is an icteric discoloration of the mucosæ and if bile pigment appears in the urine.

In **catarrh of the large intestines** diarrhea is rarely absent; symptoms of colic, however, may be absent or quite mild; the intestinal sounds are lively and loud flatus is abundantly discharged from the rectum.

Catarrh of the rectum is characterized by frequent, scanty defecation with tenesmus; the feces contain numerous shreds of mucus, which coat the lumps and masses like membranes; the feces are also frequently streaked with blood. On being introduced into the rectum, the finger or hand feels convulsive contractions of the sphincter muscle which later on relaxes, and this leads to prolapse of the rectal mucosa which feels hot and slippery; the introduced finger or hand becomes covered with mucus or blood. By the rectal speculum a markedly reddened mucosa becomes visible.

The symptoms of sugar and molasses disease were as follows: Restlessness, profuse perspiration, weakness in the posterior parts, periodic absence of peristalsis, retained urine, acceleration of the pulse, increased thirst, extreme filling of the urinary bladder, a reddish to coffee brown discoloration of the urine, filling of the large intestines with masses of soft feed, soft, foul-smelling feces covered with mucus, and also, at the height of the disease, frequent lying down, profuse perspiration, especially in the posterior parts of the body, and sometimes symptoms resembling founders. Following suitable treatment the animals were always temporarily relieved, but after a few hours the disturbance again increased in severity and the animals died with symptoms of heart weakness about 24 hours after taking sick. Only light cases gradually recover after the feeding of sugar was discontinued. (Sustmann, Eber, Bergemann.)

The turnip disease of horses, according to Sustmann, begins with severe colic, which is followed by a greatly depressed appetite, increased peristalsis, somewhat accelerated pulse, and the passage of soft to thin feces streaked with blood. Improvement takes place in 1 to 2 days, followed by recovery, though a certain weakness of the posterior parts is noticeable up to 8 days.

In the disease in horses caused by sliced turnips the symptoms consist of obstruction of the esophagus, acute dilatation of the stomach and acute gastro-intestinal catarrh, while in some cases where bronchopneumonia is present gangrene of the lungs may supervene (Menneking).

The acute gastrointestinal catarrh in horses caused by feeding rye is followed by founders with edematous swelling of the skin of the coronet and heel, sometimes reaching to the carpal or tarsal joint. Death rate 10% (Naumann).

The disease caused by mixed feed is characterized by extreme restlessness, constipation, fecal balls covered with mucus, decreased peristalsis, pulse accelerated up to 80 per minute, fever up to 40.8° C. (105.5° F.), frequent passage of urine, serous to mucous discharges from the mucous membranes of the head, depression and symptoms of founders. Eating and drinking are suspended for a short time only. Constant recovery follows in 12 to 72 hours.

In bran-poisoning in horses Nevermann observed colic, distention of the abdomen, inhibition of peristalsis, weakness in the back even to such an extent that the animal could no longer get up, constipation, evacuation of foul-smelling liquid feces after the administration of laxatives, fever up to 40.8° C. (105.5° F.), accelerated pulse, sometimes slight erosions on the back on the tongue, painful swelling of the scrotum and prepuce in stallions, and intense thirst. Autopsy showed peritonitis, decided swelling of the intestinal mucosa with punctiform hemorrhages, especially in the large intestines, and firm swelling of the spleen.

An exclusive diet of horseflesh frequently led to watery discharges from the intestines, without other grave symptoms, shortly after feeding, or at most within a few hours.

In cases of pyocyaneous bacillosis in young pigs taking a less rapid course, exanthema with the formation of scabs, nasal catarrh, pharyngitis and pneumonia may also develop under certain conditions.

Enteritis anaphylactica in dogs is characterized by a dropping discharge of bloody fluid from the anus and the usual symptoms of intestinal catarrh. Clinically and anatomically it often resembles spontaneous hemorrhagic gastro-intestinal catarrh of dogs.

Course. Mild cases last from two to three days, and then the slight disturbances disappear. There are frequently short attacks of restlessness; after the expulsion of gases there are sev-

eral fluid stools, then marked improvement occurs leading to rapid recovery. In a majority of cases, however, the disease is of longer duration; it lasts about a week and the animals are weak for some time, even after the disappearance of the symptoms. If the intestinal catarrh is more severe, the animals become more and more exhausted in consequence of obstinate diarrhea and a fatal issue may occur in the second or third week. If the disease lasts still longer it terminates in chronic catarrh of the intestines. There are also very grave cases which show profound prostration and grave intestinal disturbances from the start and which end fatally within three to four days. Cases of this kind are observed particularly after the ingestion of spoiled feed or putrid meat and they must really be classified as deeply penetrating inflammations.

A frequent complication of intestinal catarrh is gastric catarrh; its symptoms generally precede, but occasionally follow, those of intestinal catarrh. Acute intestinal catarrh may also be followed by albuminuria or even by parenchymatous or diffuse nephritis; in horses diffuse superficial pododermatitis may make its appearance. An urticaria-like eruption is not infrequent, and in exceptional cases clonic convulsions of the abdominal muscles, the diaphragm or other muscles are observed. Bacteria which have been absorbed and are eliminated by the kidneys may give rise to catarrh of the urinary bladder.

Diagnosis. The diagnosis meets with difficulties only in the absence of diarrhea, but a correct diagnosis is suggested by lively intestinal sounds, in connection with symptoms of colic, possibly with icterus, and by the fact that the general disturbances and those of the pulse are usually not considerable.

According to the above definition of intestinal catarrh, the presence of diarrhea does not under all conditions indicate that inflammatory changes have taken place in the intestinal mucosa, since the diarrhea may be of purely functional origin. With certain restrictions the macroscopical or microscopical detection of mucus in the feces may be accepted as a positive indication of intestinal catarrh, though the admixture of mucus in the feces may indicate the presence of but few catarrhal changes. An exception is noted in those cases in which, because of the presence of hard fecal matter in the rectum or in occlusion of the intestinal tract, the mucosa of the rectum is incited either mechanically or by reflex action to excrete large quantities of glistening, whitish masses of mucus (especially frequent in the horse). On the other hand, the absence of mucus does not exclude a catarrh of the small intestines, since the mucus might have been digested or have been overlooked in the feces. Inflammation of the intestines usually leads to grave disturbance of the general condition from the start, weak and frequent pulse, obstinate lack of appetite, and the impossibility of influencing the disease favorably by treatment.

Since intestinal catarrh may be due to acute infectious diseases and to intestinal parasites, these conditions must be considered in the diagnosis.

For the detection of mucus in the feces of dogs Jakob makes an enema of lukewarm water (100 to 200 cc.) and after 5 to 10 minutes siphons it into a white dish by lowering the infusion funnel and inspects it for the presence of mucoid admixtures. In case the macroscopical examination is negative a small quantity of the fecal material is examined under the microscope after the addition of acetic acid. The mucus will become visible as streaks.

Prognosis. Primarily uncomplicated intestinal catarrh is dangerous only in very young or very old animals; in these it may occasionally lead to death; as a rule the disease ends in recovery and rarely leads to chronic catarrh. Of unfavorable prognostic significance are elevation of temperature, very severe pains, marked prostration, obstinate diarrhea; these symptoms point to secondary inflammation of the intestines.

Infectious gastro-intestinal catarrh of hogs usually disappears, under purely dietetic measures within one to four days, sometimes only towards the end of the first week. Fatalities do not occur as a rule; Aronsohn, however, reported a mortality of 1 to 2%.

Treatment. The main points in the treatment of acute intestinal catarrh are the establishment of favorable hygienic conditions and the proper regulation of diet. The sick animals should, above all, be kept quiet in a warm, dry place where they can, if necessary, be protected against loss of heat by being covered with blankets. The selection of such a place is particularly important in young and small animals, including rabbits and fowls. The feeding ought to be done in the most cleanly manner and the feeding vessels should be cleansed scrupulously before each meal. The parts of the body which may become soiled in defecation must be cleansed, the feces must be removed promptly and frequently from the neighborhood of the sick animals, and an admixture of feces with feed must be strictly prevented. The buccal cavity of the animals should be wiped out several times each day with pure or somewhat acidulated water.

The noxious feed had better be discontinued or its noxious effect at least ameliorated by proper additions. Thus diarrhea following the feeding of green leaves of sugar beets can be quickly relieved by the addition of other roughage, especially when hay is given as the first feed in the morning. The same is true, according to Loew, when beet-top silage is fed, if, on the night previous to feeding, it is thoroughly mixed with 8 gms. precipitated chalk and 2 gms. sodium carbonate per kilogram of the silage and then firmly pressed together, after the addition of a little water if necessary. The noxious effect of turnips and turnip chips can be partly overcome by liberal additions of chopped roughage (also by previously soaking the dry chips),

by the liberal feeding of hay or by the addition of precipitated chalk. As a preventive against the sugar and molasses disease Ellenberger and Waentig recommend the proper addition of other feed (bran, turnip chips, etc.), precipitated chalk or calcium phosphate, charcoal and stock salt. When cane sugar is fed, a liberal quantity of hay and chopped roughage should also be allowed. Horses that are very sensitive to cane sugar should be fed only very small quantities of cane sugar or molasses in case such material must be utilized.

To remove the noxious effect of horseflesh, Pflueger found it very satisfactory to feed the cooked horseflesh without the broth, also to add 2½% tallow from sheep or cattle, or 10% to 20% rice with 2½% beef tallow. On the other hand, the results were not entirely satisfactory when the bones were also fed, or when 2% to 3% calcium carbonate was added.

The regulation of the diet should protect the inflamed mucous membranes against further irritation and should attempt to furnish as little culture-material as possible to the microorganisms which rapidly increase in the abnormal intestinal contents. Animals should, therefore, receive a starvation diet for one or two days, if they come under treatment at the first stage and if their state of nutrition permits of the withdrawal of food. All irritating, acrid, undigestible food must be avoided and food must in general be given only in small doses for each meal. Herbivora may receive aromatic, but not fresh, hay (perhaps scalded), grains softened in warm water, crushed oats or barley (best mixed with water and salts), flour or bran mash and, in case the diarrhea is severe, mucilaginous soups of oats, barley, linseed cakes; for ruminants in addition boiled beets and potatoes. Raw feed cannot, as a rule, be withheld entirely, since the animals will then refuse all food. Sick swine must be fed with flour, rice flour, germinated malt, linseed, bread soups, roasted oat or barley flour, roasted chestnuts, rice, sago, boiled oatmeal with fat, meat broth with the yolk of eggs or a variety of artificial foods (see page 134). After the diarrhea has been stopped, finely chopped meat or fowl meat may be added to the enumerated food stuffs or the patients may receive a mash of crushed barley, rice, etc., to which has been added some fat and salt. Rabbits should receive roasted oats, young twigs of oaks or willow, also burnt flour soup, thick flour paste with roasted rye bread. The following are adapted for fowl: millet, rice, corn and other grains, boiled or worked into a mash with the addition of powdered chalk; also roasted and finely crushed barley, and infusion of half an ounce of oatmeal boiled in one quart of water, or one part of linseed boiled in twenty parts of water; parrots do well on chocolate or on bread moistened with claret. The ingestion of water must be limited and the animals must not receive it cold, but water which has been standing for some time. In mam-

malians, especially in young animals, good results have been obtained with the administration of Yoghurt, it being assumed that the Yoghurt bacilli exert an antagonistic action upon the intestinal flora, more particularly upon the bacteria of the coli-group (Standfuss).

Since acute intestinal catarrh generally follows errors of diet, the removal of the gastro-intestinal contents is indicated; this diminishes the amount of the irritating material already in the gastro-intestinal tract, removes a culture soil for excessively multiplying intestinal bacteria and to a certain extent brings about a disinfection of the intestines. If the noxious material is probably still in the stomach then lavage of the latter or the use of emetics may be indicated. The treatment of disturbances of gastric functions, if at all present, must follow the principles laid down above (see page 131). To remove the noxious or abnormally fermenting contents of the intestines mild laxatives should be employed; the best drug is probably castor oil 250-500 gm. to horses; 500-1000 gm. to cattle; 50-200 gm. to calves, foals, sheep and goats; 50-100 gm. to hogs; 15-50 gm. to dogs; 5-20 gm. to cats; to rabbits and birds 5-15 gm. To horses and cattle castor oil is administered with the double dose of hot water or with the same or the double dose of a bland oil (oil of sesame, oil of poppies, etc.); to the dose for horses may also be added 50-70 grams of ether; this makes the oil light-fluid; for dogs and cats the oil may be emulsified with one-fifth gum arabic and five to ten parts of water or with aqua menthæ, tinctura aurantii, succus citri, etc., or in gelatin capsules (3-10 at 5 grams each); to the hog in the form of an electuary with licorice or honey; for fowls a mixture with equal parts of water should be injected directly into the esophagus with a syringe armed at its tip with a flexible tube; or small pieces of stale bread soaked in castor oil are introduced into the pharynx of fowls (Schlampp).

With calomel a laxative effect can be obtained only in dogs (0.1 to 0.4 gm.), cats (0.1 to 0.15 gm.), rabbits (0.1 to 0.2 gm.), and in birds (0.2 gm. for chickens, 0.05 gm. for pigeons). In the horse poisoning was observed after a once-repeated dose of 8 gm. (Froehner). Like castor oil, calomel should be given in one full dose. Neutral salts (magnesium and sodium sulphate) are also very effective (horses 250 to 500 gm., cattle 500 to 1,000 gm., sheep and goats 50 to 100 gm., hogs 25 to 50 gm., dogs 10 to 25 gm., cats, rabbits and birds 1 to 5 gm.). They are given whenever possible in dilute (about 5%) solution. Judging from the experience of physicians, a mild laxative effect in dogs may be obtained by the subcutaneous injection of magnesium or sodium sulphate in a 25% sterile solution (dose for man 1 cc., if necessary repeated on several consecutive days). In some cases subcutaneous injections of laxative alkaloids in fractional doses 1 to 3 times at 2-hour intervals have proved beneficial as not irritating the intestinal mucosa and acting more quickly (for

horses eserin 0.02 to 0.03 gm., pilocarpin 0.05 to 0.08 gm., arecolin 0.01 to 0.02 gm.; for other species correspondingly smaller doses: for cattle eseridinum tartaricum 0.06 to 0.08 gm. at a dose). In case of emergency barium chloride in a single dose of 0.1 to 0.15 gm. could be given intravenously to horses and cattle. The often recommended sennatin (prepared from senna leaves), although suitable for increasing peristalsis, has a laxative effect only in dogs and cats and is not without danger in cats (Hertz, Koestlbacher). It is used subcutaneously (for horses 20 to 30 gm., sheep and goats 5 to 15 gm., dogs 2 to 4 gm., rabbits 3 gm.). Follikulin (also prepared from senna leaves) is, according to Hertz, not so reliable as some of the older, well-known laxatives and has a reliable laxative effect only in cats; in other animals it can not be used at all. Hormonal (prepared from spleens; so-called Peristaltikhormon) was found by Engel, when used subcutaneously, to be a mild laxative for rabbits (5 to 10 gm.), dogs (10 to 20 gm.), swine (20 gm.), sheep (10 to 15 gm.), and cattle (30 to 40 gm.). Most animals reacted temporarily with fever (due to foreign protein). Drastic purgatives should be avoided under all conditions. The contents of the posterior portion of the intestinal tract are removed more properly by infusions, into the rectum, with lukewarm water allowed to run in under moderate pressure.

The action of disinfecting methods appears problematical, especially in the long intestinal tract of herbivora and hogs. The best disinfection of the gastro-intestinal tract is accomplished by a thorough evacuation of the contents of stomach and intestines. The following drugs, however, are much used: resorcin (10-15.0 gm., or for small animals 0.5-1.0 gm.), lysol (10-20.0 or 1-2.0 gm.), creolin (the same doses), creosote (5.0-10 gm. or 0.1-0.2 gm.), naphthalin (10-15.0 or 0.05-0.2 gm.) Especially good results have been reported with the use of creolin. Guenther used urotropin successfully in dogs (also in tablet form). Naucke as well as Reiff obtained good results from the use of mercaffein, which may be used not only subcutaneously but also per os (0.05 gm. saccharum).

According to the experiments of F. Müller calomel has a laxative effect only in dogs (0.3-0.4 gm.), cats (0.1-0.15 gm.), rabbits (0.2 gm.) and birds (0.2 for chickens, 0.05 for pigeons). Like castor oil, calomel must be given in one full dose. Neutral salts (sulphate of magnesium and sodium) are also serviceable (horses 250-500 gm. as an electuary, cattle 500-1000 gm. as an infusion; sheep and goats 50-100 gm. as an infusion, hogs 25-50 gm. as an electuary, dogs 10-25 gm. in solution; cats, rabbits, fowl, 1-5 gm. in solution). Stronger purgatives should be avoided. The contents of the posterior portion of the intestinal tract are removed more properly by infusions, into the rectum, with lukewarm water allowed to run in under moderate pressure.

In mycotic intestinal catarrh in horses copper sulphate (4 to 12 gm. daily dissolved in hot water as an electuary for several days) acts as a specific. The mycelia will disappear from the feces within a few days.

Other therapeutic methods are indicated comparatively rarely, viz., when the diarrhea persists in spite of the regulation of diet and of elimination. It would, however, be an error to use anti-diarrheal drugs at once upon the appearance of diarrhea. If diarrhea persists narcotics like opium are indicated (for horses 5-20 gm., cattle 10-25 gm., calves, foals, sheep and goats 1-3 gm., dogs 0.1.0.5 gm., cats 0.05-0.2 gm., rabbits and birds 0.05-0.1 gm.), tincture of opium about 10 times the above doses of opium (powdered); both may be used in the form of suppositories or clysmata. When the symptoms are particularly violent the subcutaneous use of morphia may be indicated, although only in horses (0.3-0.6 gm.) and dogs (0.02-0.15 gm.). Of the numerous astringents the following are most commonly used: oak bark (25-50 gm. for horses and cattle, 5-10 gm. for foals, calves, sheep and goats, 5-10 gm. for dogs and cats, 0.5-1.0 gm. for rabbits and birds in the shape of a decoction, and electuary or as pills; tannin (horses 5-15 gm., cattle 10-25 gm., calves, foals, sheep and goats 2-5 gm., hogs 1-2 gm., dogs 0.1-0.5 gm., cats and rabbits 0.05-0.2 gm., fowls 0.01-0.05 gm.); further, tannalbumin veterinarium (6-30 gm. for large, 2-5 gm. for medium-sized, and 0.1-0.5-2 gm. for smaller animals); tannoform (the same doses), tannigen (the same doses), tannalbuminate-salicylate (the same doses). These preparations are all expensive, hence they can be employed usually only in small animals. Other astringents which might be mentioned are cortex chinæ (for dogs 2-5 gm. as a decoction), bismuthum subnitricum (for horses 5-15 gm., cattle 10-25 gm., hogs 2-5 gm., dogs 0.5-1 gm.), bismuthum subgallicum (dermatol, the same doses), bismuthum tribromphenylicum (xeroform, the same doses), tannismut or bismuthum bitannicum (according to Gottschalk, for horses and cattle 15-30 gm., for dogs 2-6 gm.), gastrosan (bismuth with salicylic acid) for dogs up to 0.75 gm., goats and sheep up to 10 gm. Animal charcoal (carboanimalis, carbovent), recommended by Reisinger, frequently has a very favorable antidiarrheal effect. It should be given twice daily, suspended in lukewarm water, to horses and cattle 250 to 300 gm., suckling calves 10 to 20 gm., yearlings 40 to 80 gm., sheep and goats 20 to 30 gm., hogs 20 to 50 gm., pigs and lambs 5 to 10 gm. Bolus alba suspended in water is also recommended by Reisinger. The daily dose for horses is up to 500 gm., cattle 1,000 gm., sheep, goats and hogs 200 gm., dogs 100 gm. Both remedies may be used alternately. Calcium therapy also deserves attention, especially the administration of lime water (horses and cattle 1 to 2 liters, calves, colts, sheep, goats and hogs 1/4 to 1/2 liter, dogs, cats, and birds by the tablespoonful to teaspoonful), calcium chloride (horses and

cattle 10 to 20 gm., dogs 0.5 to 1.0 gm. daily in 5% solution), calcium carbonate, calcium phosphate on the feed or suspended in water (horses and cattle 10 to 50 gm., colts, calves, goats, sheep, hogs, 5 to 20 gm., dogs 0.5 to 5.0 gm., cats 0.2 to 1.0 gm., birds 0.5 to 5.0 gm.). Kindberg found gelatin very good for diarrhea in dogs (probably because of the calcium content). Vidal and Boissière and Charmoy saw a rapid disappearance of diarrhea after subcutaneous injection of hordeinum sulphuricum (an alkaloid prepared from the waste in the manufacture of malt) into suckling calves (1 gm.) and into yearling (5 gm.). Kreutzer used to advantage uzara (prepared from an African shrub) in calves (50 to 70 drops), pigs (10 to 20 drops) and dogs (20 to 40 drops). Holterbach praises the good action of pankrofirm (extract of pancreas coupled with milk protein), of which horses and cattle should receive 5 to 10 gm., calves and colts 3 gm., dogs 1.5 gm., on the feed. Schiebel stopped the diarrhea in colts occurring a few days after birth by the administration per os of 500 cc. of blood from the dam. The diarrhea ceased on the following day.

In catarrh of the rectum, particularly in carnivora, the internal treatment indicated should be combined with rectal injections with disinfectants and astringent drugs (argentum nitricum 1-5:1000, alum, tannic acid, sulphate of iron in 1 to 2% solutions, also starch in water, perhaps with some tincture of opium, or with the use of suppositories.

Moderate and even heat is also beneficial and reduces the peristaltic movements and in this manner acts against abdominal pain. Hence warm fomentations on the abdominal wall are indicated; also warm infusions given internally. Fowls are often influenced very favorably by hot sand baths (60-70° C.) (Klee).

To counteract the severe weakness which often comes on in young animals rubbing, warm packs, subcutaneous injections of caffeine and camphor are beneficial. Most reliable, however, for this purpose is the intravenous, subcutaneous or intrarectal injection of physiologic salt solution. This latter is best brought to body temperature, after having previously been sterilized, and is injected with aseptic precautions under the skin or into a vein. The intravenous or subcutaneous dose for large animals is 4-5 quarts, 2 quarts for medium-sized animals, ¼-½-1 quart for smaller animals.

14. Acute Gastro-Intestinal Catarrh in Young Animals.

Etiology. The gastro-intestinal organs of young, particularly of sucking animals, are very sensitive and become affected even after slight errors in diet. The epithelial covering of the gastro-intestinal tract is tender and very sensitive, the muscularis is unable to make any greater efforts; the basal cells of the

gastric glands are poorly developed, the gastric juice contains comparatively little pepsin, and the protective layer of mucus on the intestinal mucosa is lacking for several days after birth. In ruminants there is the further cause that fore-stomachs are not yet functioning and the fluid food enters the abomasum without any preliminary preparation.

Since sucklings, as a rule, receive only milk, an improper composition of the latter is generally the cause of the affection. The character of milk is rarely influenced unfavorably by a too abundant feeding of the mother animals with green feed or clover hay. Much more dangerous is the milk after insufficient feeding with watery, non-nutritious, spoiled feed. Certain component parts of the feed, poisonous, oily resinous plants, and certain drugs, particularly laxatives, find their way into the milk and exert a disease-producing effect upon the digestive organs of the sucklings. The milk of cows which receive an abundance of distillery slop or oil cakes may produce gastro-intestinal catarrh in calves.

Certain diseases of the mother animals produce changes in the functions of the mammary glands. Most dangerous in this respect are the acute infectious diseases and still more the inflammatory diseases of the udder. In the course of acute infectious diseases the milk, which is also changed in its composition, frequently contains toxins, and in mastitis certain pathogenic bacteria may coagulate the milk in the udder, or this may have undergone slimy or other changes. Hence sucklings almost invariably become sick if the mother animal suffers from acute parenchymatous or any other form of mastitis, from foot-and-mouth disease, smallpox, epizootic aphthæ, or tuberculosis of the udder. Excessive exertion of the mother animal likewise disturbs the proper secretion of milk. Artificial feed often plays an etiological rôle, due to the frequent lack of cleanliness and the use of various milk substitutes which are easily decomposed and skimmed milk with the addition of flour, bread, etc. The destruction of the milk ferments by boiling also has a deleterious effect.

The ingestion of too large amounts of otherwise unobjectionable milk often produces digestive disturbances if young animals suck only at long intervals, for instance morning and evening; or if the young animals are permitted to suck too often.

Finally gastro-intestinal disturbances may be brought about because the young animals have not been permitted to suck the colostrum which removes the meconium from the newborn. Frequently a catarrhal affection of the gastro-intestinal mucosa is due to marked cooling of the body in consequence of staying in cold, damp, drafty barns, or in consequence of sucking from a cold udder, or staying in the open air during cold weather or during rain.

Improper weaning may also produce the affection if only dry feed is given at once to the weaned young. The gastric mucosa is excessively irritated by the coarse feed and at this period the fore-stomachs do not yet function properly in ruminants.

In weaned animals the disease may also be due to the same factors which cause it in adults.(see pages 311 and 352).

Concerning the ability of the intestinal bacteria and of the pathogenic microorganisms, to give rise to inflammation, the same is to be said as for intestinal catarrh of grown animals.

Sometimes intestinal parasites may produce a catarrhal inflammation of the gastro-intestinal mucosa. Regarding intestinal catarrh in the course of infectious diseases and intestinal coccidiosis, see the respective chapters in Volumes 1 and 2.

Anatomical Changes. Post-mortem examination frequently shows rather insignificant changes such as hyperemia, a moderate amount of swelling of the gastro-intestinal mucosa; in other cases there may be small hemorrhages, swelling of the follicles, sometimes also superficial ulcerations.

Symptoms. The disease usually begins with a diminution of appetite and with a certain degree of lassitude after which febrile symptoms come on. The sucklings do not hunt up their mothers; if they are taken to her they suck little and without relish; weaned animals take little food or none at all. Sometimes vomiting occurs and a marked improvement is then noticeable.

After a short time diarrhea comes on and this remains the prominent feature of the clinical picture and it may even be the very first symptom. The feces become thin and mushy, finally perfectly fluid and are sometimes voided in a stream at short intervals, under tenesmus. At this time the feces are yellowish or yellowish-gray, distinctly acid in reaction and of a disagreeable, penetrating, sour smell; they contain white or gray flocculi, lumps (fat-droplets, fat-crystals and bacteria), sometimes bloody streaks, or they are even uniformly reddish. The animals lie curled up on the floor or stand up with their backs curved; the legs are drawn under the abdomen and the patients exhibit manifestations of abdominal pain which is generally severe.

The abdomen appears either drawn in and sunken in at the flanks or it is, on the contrary, bloated moderately; the latter condition is seen particularly in ruminants. The intestinal sounds are sometimes exceptionally loud and rumbling. Palpation in the region of the stomach elicits tenderness rarely if at all (the abomasum in ruminants).

If diarrhea is obstinate debility and emaciation rapidly appear. The appetite is now entirely lacking, while thirst may be intense; the eyes sink in, the fur becomes scrubby, the skin is dry or covered with a clammy sweat, the whole body emits a dis-

agreeable nauseating odor. Finally the animals are hardly able to stand on their feet; they lie apathetically on the floor, the peripheral portions of the body become as cold as ice, involuntary movements of the bowels occur and death takes place in syncope or with convulsions.

Course. In a majority of cases the disease ends in recovery. The diarrhea stops after a certain time, occasionally after a few hours; the appetite improves, the animals become more lively and generally recover rapidly, even in those cases where the affection has lasted for some time. Grave cases lead to death in three to five days, occasionally, however, they drag along for several weeks and a catarrhal pneumonia is then usually the terminal affection. Aphthous stomatitis is a frequent complication in lambs.

Diagnosis. The disease may be confounded with dysentery of sucklings; the latter, however, always attacks the animals immediately after birth or at the utmost during the first few days of life; it takes a much more unfavorable course and betrays clearly its infectious character, while simple gastro-intestinal catarrh appears sporadically, and even if there are a number of cases, one is able to demonstrate the common external factor in its production.

The elimination of other infectious diseases, intestinal coccidiosis and helminthiasis should also be attempted.

Prognosis. The younger the sick animals, the longer the disease lasts and the less favorable is the prognosis. The latter also depends upon whether it is possible to remove the noxious causative factors.

Treatment. The first attempt at treatment must be the regulation of the diet of the sick animals; if they are sucklings, the diet of the mother animal must likewise be attended to and errors of diet or external unfavorable conditions have to be corrected speedily. In case the young animals have to be brought up by hand, it is advisable to feed them with pasteurized milk, formalin milk (1 to 25,000) or perhydrase milk. The detrimental effect of feeding milk may be diminished by the addition of lime water (a teaspoonful to a quart of milk); its often markedly bloating effect may be removed by the addition of boiled oatmeal. Lafitte saw unfavorable results from the exclusive use of fresh whey. Sucklings should be allowed to feed three or four times per day and if necessary should be fed by a healthy foster-mother. The diet of weaned animals must be regulated in the same manner as is recommended in intestinal catarrh of adult animals (see page 178). The barn should be kept uniformly warm and the place where the young animals are must be cleansed very thoroughly. The teats of the mother animals should also be cleaned before the young are allowed to suck.

The medicinal treatment is similar to that used in dysentery of the newborn (see Vol. I) or to that used in gastro-intestinal catarrh of adult animals (see page 179).

In calves favorable results have been obtained by the administration of salicylic acid with tannic acid āā 1-2 gm. three times a day in camomile tea. Schwarzmeier recommends for foals tincture of opium (up to 5. gm. pro dosi) with tannic acid in whiskey; Schley, for calves, a ½% solution of nitrate of silver. Sometimes washing out of the rumen (see page 10:) may become necessary (Imminger). Eber has brought about a cure in two calves, after collapse had occurred, by the subcutaneous injection of two quarts of physiologic salt solution (Na Cl 0.8%, carbonate of sodium 0.25%). The absorption of this solution goes on very rapidly, especially if aided by massage. A 4% solution of glucose is probably still more effective. Kronacher had similar favorable results with the rectal application of physiologic salt solution.

15. Catarrhal Intestinal Colic. Enteralgia Catarrhalis.

(*Einfache Reiz-, Krampf-, rheumatische* or *Erkältungskolik* [German].)

What is generally called rheumatic or convulsive colic is a mild, short attack of acute intestinal catarrh with colicky pains, mostly as a form of functional disturbance of the intestinal tract.

If a special chapter is here devoted to this affection, this is done because the affection is frequently treated separately in veterinary literature as a form of colic. The French writers include the disease in the great group of "*congestions intestinales*," to which also belong the disturbances due to thrombosis of the mesenteric arteries. The term "convulsive colic," "*Krampfkolik*" (German), appears objectionable because all genuine colicky pains are due to convulsive contractions of the intestines.

Occurrence. Symptoms of colic in the horse undoubtedly are due most commonly to a transitory acute intestinal catarrh. Thirty-five per cent of the colic affections seen in the Budapest clinic are due to this affection and it is the most frequent type of colic among breeding and cavalry horses. Cattle and hogs are likewise sometimes subject to this disease.

Etiology. The causative factors are the same as in acute intestinal catarrh (see page 17( ). In a certain proportion of cases external or internal cooling of the body plays a rôle; (drenching, standing in cold weather in the open air when heated, the ingestion of excessively cold water or of cold frosted feed). Catarrh from these causes is, however, rarer than catarrh due to errors of diet. The loud intestinal sounds which

are considered characteristic for the disease, the socalled "cramp sounds," clearly point to lively fermentative processes in the intestinal tract with an increased formation of gas; the disease occurs just as frequently during the warm season, when the animals are less exposed, as during cold weather. Cavalry horses, which, as a rule, receive little rough feed and which in consequence often feed upon the bedding straw, frequently suffer from the disease after the ingestion of mouldy straw, especially after the fall manœuvers when the horses are not in their usual good nutrition and therefore are more apt to eat the straw nsed for bedding. The number of attacks of colic increases with the degree in which work is imposed upon military horses (Hoffmann). In cattle, particularly in working oxen, catarrhal intestinal cramps are often caused by the ingestion of large amounts of water similarly as in faulty diet, especially when green feed is again allowed (so-called May disease, May colic).

Pathogenesis. The irritating substances (gases, fatty acids) or too much cold water cause convulsive contractions in various portions of an irritable intestinal wall, which vary in duration. These convulsive contractions then cause the colicky pains (Bauchzwicken, Bauchgrimmen [German]). After the abnormal intestinal contents have been voided, the convulsive contractions cease, but the peristalsis for some time remains more lively because the intestinal mucosa is in a catarrhal condition.

Symptoms. Horses are subject to symptoms of colic which are usually violent; the individual attack lasts from five to fifteen minutes and the animals appear well during the intervals. The intestinal sounds are usually intensified and more frequent, sometimes continuous and occasionally audible, not merely over the cæcum but likewise over the remainder of the intestines. Defecation may be perfectly normal, that is, if only the small intestines are affected, or it occurs more frequently and the feces are soft and contain undigested particles of feed; sometimes there are short attacks of diarrhea. In **cattle** severe attacks of colic come on suddenly, the abdomen is occasionally somewhat enlarged and the left flank depression is somewhat filled out, although bloating is absent. After half an hour diarrhea sets in and with it a gradual diminution of the pains; the animals usually appear perfectly recovered after a few hours.

Hogs become very restless in consequence of catarrhal intestinal pains; they lie down frequently, roll, groan, sigh, or cry out aloud. After the expulsion of flatus and after several defecations they become quiet.

Course. In the course of mild cases the attacks of pain last only a few quarters of an hour; the affection rarely extends over

ix hours, but even after the symptoms of pain and restlessness ıave disappeared, the intestinal sounds persist for some time. The course is favorable almost without exception; only very 'arely do excessively strong intestinal contractions lead to 'olvulus.

Diagnosis. The appearance of abdominal pain in parxysms, the absence of an increase of the abdominal circumfernce in the horse, usually also in cattle and swine, in connection 'ith a lively peristalsis with commonly more frequent defecaion, the absence of general disturbances, the negative result of n exploration per rectum are characteristic for the affection nder discussion. Embolism of smaller intestinal arteries ocasionally also causes abdominal pain. A differential diagnosis s only possible, and then not always, if the history shows peridic attacks of abdominal pain without any external cause, with normal character of the feces, and when rectal exploration emonstrates thrombosis of the mesenteric artery, in which case ne may, of course, assume an embolic closure of some small instinal vessels.—Abdominal pains due to uterine contractions, 'hich in mares come on towards the end of gestation and are ue to energetic fetal movements, do not lead to an intensificaon of the intestinal sounds and the fetal movements can easily e perceived.—Strangulation or invagination in cattle can e excluded from the history and the findings.

Occlusion of the urethra by urinary calculi in oxen, postitis, abdominal abscesses, etc., can be eliminated by reason of he continuation of the discharge of urine (wetting of the hair round the prepucial orifice, soaking with urine of a sack astened in front of the prepucial orifice), absence of pulsating novements in the urethra in the perineal region and excessive istention of the urinary bladder (Schwind).

Treatment. Warm applications upon the abdomen and recal injections with warm water have a tendency to stop the ramps of the intestines. Warm applications may be made in uch a manner that two sacks are sewed together, dipped in 'ater of 40° C., placed upon the abdomen and covered with a dry lanket. The application must be renewed every ten minutes. f in horses the abdominal pain is very intense morphine (0.3-0.5 m.) may be given subcutaneously or chloral hydrate per recım. To expel the decomposing intestinal contents rapidly mild ıxatives may be given which are not liable to lead to cramps, neutral salts, castor oil, combined with ether for horses); the iet should be regulated as in acute intestinal catarrh (see page 78).

For the prevention of intestinal colic a regular diet is of nportance. Military horses should receive proper quantities f hay, so that they need not eat their bedding; in consequence ıey will make better use of their grain-feed. The bedding hould be good and dry (Hoffmann).

Abdominal Pain in Uterine Contractions. Highly bred mares frequently show symptoms of colic in the advanced stages of gestation, especially about the 8th month of pregnancy and during the last weeks before parturition, these depend upon a mechanical irritation of the uterus by the moving embryo and upon uterine contractions caused by it. Predisposing causes are changes in position of the embryo, external dull force to the abdomen, overheating followed by the ingestion of cold water, the ingestion of spoiled feed.

The symptoms are similar to those of catarrhal intestinal cramps, but the hand placed upon the abdomen may feel the kicks of the embryo and they may become visible on the abdominal wall. The affection lasts from 1 to 3 hours and then disappears suddenly; in some mares the painful attacks recur within short intervals, sometimes every second day, even every 6 to 8 hours, especially shortly before parturition; (Schleiffer Vet. 1894, 229).

For treatment subcutaneous injections of morphine, also the internal administration of camomile tea are indicated.

16. Chronic Intestinal Catarrh. Catarrhus intestinalis chronicus.

(*Enteritis catarrhalis chronica.*)

Similarly as in acute intestinal catarrh, long-continued or permanent purely secretory or motor disturbances of the intestinal tract without chronic inflammatory changes in the mucosa must also be included temporarily in veterinary medicine under the term chronic intestinal catarrh.

Etiology. Primary chronic intestinal catarrh, which is comparatively rare, usually arises from an acute intestinal catarrh if the latter has lasted for some time or if the inflammatory influences have acted for a considerable period upon the intestinal mucosa. The causative factors are the same as those of acute intestinal catarrh (see page **170**), the most common cause, however, is improper feeding and the continued ingestion of improper or spoiled food.

According to the statements of Dieckerhoff, there occurs in foals, also in cattle, sheep, swine and goats, during the first two years of life, a chronic disturbance of the function of the intestinal mucosa, which leads to progressive emaciation and frequently to death. Dieckerhoff has named this disease "*Darrsucht*" (German) or Tabes intestinalis (intestinal wasting away) and has separated it from the disease Tabes mesaraica. (See Vol. I.) The etiology of the disease is unknown, its causes are probably various emaciating internal diseases. According to Glage, this Tabes intestinalis in foals is in reality an infectious sclerostomiasis (q. v.).

Dawson has described a disease observed in the United States under the name of Infectious Asthenia in chickens two to six months old, which is caused by a bacterium of the colon group (bacterium astheniæ) and characterized by a profound duodenal catarrh. The same disease has occasionally been observed in Germany by Kitt.

Some of the cases described by Dieckerhoff and Harms as chronic

gastric catarrh of cattle, and a disease described in France as "diarrhée chronique," "boyau tendre," "dysentérie," should more properly be classified as "enteritis paratuberculosa (Bang). (See Vol. I.)

More commonly chronic intestinal catarrh is a secondary affection. Such secondary chronic intestinal catarrhs are seen in chronic passive congestion of the intestines due to chronic diseases of the heart and liver, very rarely in certain cases of thrombosis of the mesenteric artery, in chronic infectious diseases (tuberculosis, pyobacillosis [Grips, Glage & Nieberle]), chronic hog erysipelas (Eisenmann) and in chronic constitutional diseases. Intestinal worms likewise frequently produce the picture of this disease. The cause of enteritis profunda chronica cirrhoticans eosinophilica with simultaneous thickening of the wall and ulceration in the stomach of cattle described by Joest is unknown.

Pathogenesis. The pathogenesis differs from that of acute intestinal catarrh (see page 172), in so far as the continued injurious agencies give rise to lasting alterations in the mucous membrane, especially proliferation and atrophy of the connective tissue, which may, in its turn, be followed by atrophy of the glandular layer and even by atrophy of the entire intestinal wall. *The diminished irritability of the affected portions* of the intestine influences the propulsion of the intestinal contents unfavorably; if then the absorption is not interfered with, the contents may become inspissated, but they may also become disintegrated because of a rapid multiplication of the intestinal bacteria. Owing to these processes, irritating substances are developed in large amounts which stimulate increased motions in other portions of the intestine, and also an increased secretion of liquid; after the disintegrated intestinal contents have been discharged, the original state is restored. In many cases the contents remain thin fluid, if the absorption suffers permanently or if a persistent secretion of fluid takes place into the gut, in which case the disintegration is promoted. The disturbance of digestion and of absorption being lasting in diffuse catarrh, especially with respect to the absorption of fats, but also that of carbohydrates and of proteins, the nutrition of the body cells suffers more and more, if the small bowel or, in herbivora, the cecum is involved.

Anatomical Changes. The intestinal mucosa usually appears dark purplish to brown red, sometimes slate colored, in *consequence of the presence of numerous blackish points, and* thickened, while the glands of the propria are enlarged, tough, or changed into cysts. Sometimes, however, the mucosa is thin, pale, and shows depressions in the place of the solitary and agminated follicles (socalled areolation). Occasionally there are formed round, thick but shallow, later on deeper or confluent irregular ulcerations with undermined and tough margins.

After healing, these ulcers leave radiating cicatrices. After perforation of such ulcers purulent ichorous peritonitis occurs in their neighborhood, and the affected loop of intestine becomes adherent to neighboring loops, to other abdominal organs or to the abdominal wall.

The pitlike depressions and cheesy plugs on the ileocecal valve of pigs have no pathological significance according to the investigations of Joest and Liebrecht and Finzi. According to Finzi, they are found in almost all hogs ranging in age from 7 to 18 months. They represent glands of Lieberkuehn that have become enlarged or filled with accumulated gland secretions and leucocytes, hard, grayish-white or grayish-yellow cheesy masses containing only intestinal bacteria, but considered by Finzi as partly due to ulceration. The cheesy contents can be squeezed out in the form of a plug. These changes must not be mistaken for hog cholera.

In asthenia of chickens the intestinal contents appear to be only mucous, the duodenal mucosa is reddened at its prominent points and the cadaver is much emaciated.

Symptoms. When chronic intestinal catarrh has existed for sometime, the animals are listless and their ability to work is much diminished; later on their condition becomes very poor. The abdomen is drawn in. Defecation occurs at irregular intervals, constipation lasting for several days alternates with violent diarrhea; sometimes the feces are dry and they are dropped after considerable intervals, at other times they are thin, mushy or fluid, containing poorly digested particles of food, shreds of mucus, lumps of pus, and they are sometimes streaked with blood; they are often very fetid. Occasionally masses of mucus without any feces are voided.

From time to time, particularly when bloating is present, large amounts of intestinal gases are expelled from the rectum. Peristalsis is absent during constipation, very lively during diarrhea. Colicky pains are rare, when they occur, they usually precede diarrhea or appear while it lasts. During lack of appetite in herbivora the urine often becomes acid; but Friedberger & Fröhner as well as Albrecht usually found an alkaline reaction of the urine under these conditions. Bauer found an increase in indican.

According to Dieckerhoff, the symptoms of intestinal tabes of young animals, are diminution of appetite, partial constipation, occasionally diarrhea (Krönig), emaciation progressing to the point of cachexia, and leading in three to six months to death unless something can be done by a proper change of diet.

In infectious asthenia of chickens one sees listlessness, depression, progressive emaciation in spite of greedy feeding, pale discoloration of the comb and of the wattles, of the throat and ears, and slight constipation. In the further course of the disease the appetite becomes variable. Finally, the cachectic animals succumb after the disease has lasted about three months.

Course. Chronic intestinal catarrh may last for months or even years and it may lead to complete exhaustion of the sick

animals, after an edema has developed on the extremities and in the lower abdominal region. Death takes place from marasmus. The disease usually takes this course in old, poorly nourished, hard worked animals.

Diagnosis. Progressive emaciation, occasional attacks of diarrhea, which rarely last continuously for weeks and months, the presence of undigested particles of feed in the feces are signs upon which the diagnosis rests. As to the localization of the affections the same rules hold good as are laid down for acute intestinal catarrh (see page 175). Lumps of pus in the feces point with great probability to ulcerations.

Considering the fact that chronic catarrh of the intestines is usually a secondary affection, one should always carefully examine all organs and look into the method of feeding in order to find out the primary disease. As such, one must think of chronic infections diseases, metabolic diseases, helminthiasis, chronic disease of the liver, chronic uremia, and in horses disturbances of mastication in consequence of anomalies of teeth.

The disease called "Darrsucht," according to Dieckerhoff, may be recognized from the fact that the improperly fed young animals of one herd or flock all become more or less sick and emaciate progressively. Tuberculosis has sometimes to be excluded by a properly conducted tuberculin test.

A diagnosis of infectious asthenia of chickens is suggested by the fact that the sick animals are all two to six months old, that cachexia develops progressively in spite of a good appetite, that the course is quite chronic and that there are no pronounced anatomical changes. The differential diagnosis must always consider the possibility of helminthiasis.

Prognosis. Chronic intestinal catarrh, whether primary or secondary, must always be looked upon as a serious affection which will often resist the most careful treatment for a loug time or even permanently. The prognosis is particularly unfavorable in old animals and when a proper regulation of the diet is impossible. The prognosis of secondary chronic intestinal catarrh depends upon the primary underlying disease. There is no hope of recovery in "Darrsucht" after cachexia is once present; young animals three to four months old generally succumb to this disease. Chickens affected by infectious astenia generally die.

Treatment. A rational diet (see pages 131 and 178) offers the best means of preventing the development or to stop the progress of chronic intestinal catarrh. The sick animals should be taken out daily and should be used for work only moderately. Animals attacked by "Darrsucht" may recover in one to two month if they are pastured, provided that the disease has not progressed too far.

To regulate defecation, castor oil and the neutral salts are indicated, among the latter particularly Carlsbad salt. The desired effect can, however, only be attained if the salts are administered to the animals one-half to one hour before feeding, and in moderate doses (130-500 gm pro dosi, or of the 5% solution, tablespoonful doses). This course must be continued for a long time and kept up for some time after recovery. With the salts may be combined bitters (gentiana, calamus, rheum, oak bark). Constipation must be counteracted by mild laxatives, diarrhea by astringents and mucilaginous medicines. Good results have been brought about in horses by the systematic administration of creolin. In a horse with diarrhea and abnormal gas formation, S. Schmidt obtained healing in two weeks, by daily doses of 30 gm. of salicyltannarabin, and Holterbach by the administration of pankrofirm for 3 weeks (see page 183) in yearlings with obstinate digestive disturbances.

A good result may as a rule be expected only in primary catarrh and while the general condition is still fairly good. The authors again point out the possibility that intestinal worms may be the cause of the trouble; cases which have been treated for a long time without any success at all have yielded in a short time, after an antihelmintic treatment was instituted.

Against infectious asthenia of chickens, Klee recommends the administration of castor oil (in teaspoon doses) or of calomel (0.01-0.05 gm.) until a marked laxative effect becomes manifest. The birds ought to receive further, daily twice per head, 1-2 gm. of the following mixture: Fennel, anise seed, coriander, bark of quinia, each 4 gm.; powdered root of gentian and ginger, each 7.5 gm.; sulphate of iron 2.0. As drinking water the fowls should have a watery solution of sulphate of iron and sulphuric acid (1000:2:2). The healthy animals must be separated and the coops must be disinfected.

17. Membranous Enteritis. Enteritis mucomembranacea.

(*Socalled "Darmkrupp"* [German] or *Croupous enteritis; entérite cöueneuse, ou pseudomembraneuse ou mucomembraneuse* [CADÉAC] [French].)

Membranous enteritis is characterized by a peculiar, generally superficial, inflammatory process of the intestinal mucosa, with the formation of pseudomembranes, which are composed almost exclusively of mucus.

Occurrence. This rather rare affection is observed most frequently in cattle, according to Keilgaard quite frequently in horses, rarely in sheep (Clavel, Lafosse). Among cattle, young, well nourished and pregnant animals are affected preferably if after winter feeding they are pastured during cool weather on luxuriant meadows.

Etiology. The nature of the affection suggests infectious material as its cause, while external influences, such as the ingestion of too cold water or the feed supply play a rôle as predisposing factors. Nothing definite can be said as to the nature of the supposed infectious agent. The disease has been observed frequently after the ingestion of rotten potatoes or beets or of absolutely fresh feed as well as following the liberal feeding of chops. In the horse Rogers ascribes it to an increase in the production and retention of oxalic acid in the blood (oxalemia) during metabolism.

Whether an occlusion of the intestines or the lack of peristalsis, due to intestinal bacteria, may cause an accumulation of mucus cannot at present be decided. It is, however, a fact that abundant thick masses of mucus collect in the rectum

of horses, similar to croupous membranes, if an internal occlusion, situated between rectum and stomach, prevents pristalsis of the rectum. In this connection the observations of physicians on artificial enteritis membranacea following irrigations with tannin, alum and silver nitrate, the use of strong astringents, long-continued misuse of laxatives and enemas (Mohr and Staehelin) deserve to be mentioned. Physicians assume, by the way, that the affection is frequently due to a general nervous condition (hence the name *myxoneurosis intestinalis membranacea*).

Anatomical Changes. As a rule the jejunum, cecum and colon are uniformly changed; however, the small intestine or on the other hand the large intestine alone may be the seat of pathological changes. The intestinal contents are thin fluid and dirty discolored, mixed with blood in grave cases and very fetid, floating in it are found yellowish to brown, tough, elastic membrane-like or tubular, cylindrical masses. Similar masses are found adhering to the mucosa. The tubular or cylindrical deposits are usually from 1.5 to 3.5 feet long, but they may exceptionally be as long as 35 feet (Arnal). They are composed of several layers, the membranous substance alternating with layers of darkly stained fecal matter. The intestinal mucosa shows reddening in grave cases, also hemorrhages, it is somewhat swollen. In the horse one usually finds changes only in short portions of the large intestine and the pseudomembranes are characterized by their small dimensions.

The microscopic examination of the pseudomembranes deposits show the presence of numerous granules in an exceedingly fine reticulum (Gurlt). Weigert's fibrin stain never shows any fibrin in the cases examined, and the proper stains demonstrated the presence of mucin (Johne). Chemical tests made by Lassaigne showed that the pseudomembranes consist almost exclusively of condensed mucus; the same results were obtained by Clément who investigated membranous enteritis in horses. Hey frequently found fibrin (staining according to Weigert and with thionin) only in the intestinal discharges of horses suffering from impaction colic.

All investigations made have failed to furnish any proof that membranous enteritis is a genuine diphtheritic inflammation of the intestinal mucosa, but they have rather shown that we are dealing with a peculiar purely catarrhal process with an overproduction of mucus, analogous to the membranous catarrh of the stomach of man (Nothnagel). The preferably catarrhal character of the process does, of course, not exclude an occasional deeply penetrating inflammation.

Symptoms. The disease usually begins with disturbances of digestion, mild attacks of colic and slight pressure upon the rectum. One also observes muscular tremor, reddening of the conjunctivæ and an acceleration of pulse and respiration. The secretion of milk is suppressed. The attacks of colic last only 12 to 15 hours. After five or six days, rarely somewhat later, colicky pains recur, the animals walk around restlessly, turn their heads to look towards the abdomen, kick with their feet and drop feces with signs of tenesmus. These are thin fluid and fetid, mixed with gray or yellowish-white membranous, tubular or cylindrical masses of variable, sometimes of considerable length. After one to two days the symptoms suddenly disappear; however, the feces remain fetid for several days

and are mixed with mucus; then the animals recover completely. The pseudomembranes may reappear.

Deviations from this clinical picture occur in two directions. In very mild cases animals previously apparently quite healthy, very unexpectedly drop such membranes as described, perhaps with symptoms of a little restlessness, then thin fluid feces, mixed with mucus, then they soon get well again (Lafore, Reynal, Combe). There are, however, on the other hand, cases which run from the start a course with the symptoms of a grave enteritis or these symptoms may develop later on and profuse hemorrhages may occur in the further course of the disease. In cases of this kind there is great prostration, complete lack of appetite, high fever, the pulse cannot be felt, the peripheral parts of the body feel cold as ice and the fluid and fetid feces are more or less hemorrhagic.

In horses, the disease likewise usually begins with restlessness; this soon disappears and the animals then stand apathetically and stagger on standing and walking. In other cases colicky symptoms are at first absent, the disease begins with a diminution of the appetite which soon disappears completely. There is often fever (39.5°-41° C.) and always an acceleration of the pulse. In the further course of the disease, colic appears and the feces are profusely covered with mucus; grayish-white, or white opaque, structureless or curled-up long strands or membranes of mucus may also be present free in the intestines or they may be voided with the feces. The disease always ends in the recovery of the animals after a few or several days.

Course. The whole course usually lasts from 8 to 12 days, it may be somewhat shorter or on the contrary longer; in the latter case, exacerbations occur during the voiding of the pseudomembranes and remissions at other times. The disease usually ends in recovery; a fatal issue occurs in only such cases as are complicated with grave intestinal inflammation or intestinal hemorrhages; in these the animals may succumb after four or five days. Very exceptionally there may be a fatal obstruction of the intestines by the accumulated pseudomembranes.

Diagnosis. The true character of the disease can be recognized only after the appearance of the pseudomembranes; before this occurs it cannot be distinguished from other inflammatory processes of the intestinal tract. The pseudomembranes have a typical appearance and can easily be distinguished from pieces of tendons that are accidentally taken up with the feed, from tapeworms, loops of intestines and other objects.

Treatment. The expulsion of the pseudomembranes and of fetid feces may be hastened by the administration of alkalies, especially the laxative salts (see page 180); later on mucilaginous mixtures and astringents are indicated, similar to those recommended in other forms of intestinal inflammation (see page 181). The regulation of the diet must follow the same principles as are laid down in the treatment of acute intestinal

atarrh (see page 178). In a case following infectious pleuropneumonia in a horse Kuntschik obtained rapid favorable action of arsinosolvin (in solution 3:20, subcutaneously).

Enteritis Pseudomembranacea of Cats (croupous enteritis of cats). This is a disease evidently peculiar to cats. According to Zschokke, who first described it, it is most common among young cats and appears epizootically *during winter and spring. Cases of enteritis pseudomembranacea* have been observed also by Kitt, and more recently by Schmul, who studied the histologic changes of the disease.

Zschokke believes that the probable cause of the disease is a virulent variety of the colon bacillus, and this finding has been substantiated by Reichel and Mumma, who discovered a paracoli bacterium (paratyphoid bacillus) as the cause. Feeding of the culture caused a transient intestinal catarrh and twitching of the muscles in a young cat, and it was pathogenic for rabbits and guinea pigs. Schmul believes that several kinds of baeteria are concerned in the causation of the disease.

The most important anatomical changes consist in an increase in thickness, diminished elasticity and a greater transparency of the wall of the small intestines; in about one-half of the cases there are deposits on the mucosa; the latter, however, is almost always reddened and swollen. The contents of the small intestines are of a milky turbidity, while the contents of the large intestines are frequently mixed with blood.

The histologic examination shows that the intestinal villi are markedly contracted, the tissues of the intestinal wall are edematous, there is frequently necrosis of the villi, and this may extend even into the serosa. There is also a moderate leucocytic infiltration and likewise moderate hemorrhagic extravasation.

The pseudomembranes are, according to Zschokke, composed of a mixture of epithelia, leucocytes and fibrin threads, but according to Schmul, they are mostly made up of bacteria and do not give either a distinct mucin or a fibrin reaction.

The mesenteric lymph glands usually show acute swelling; the liver and the kidneys are congested.

The symptoms of the disease consist in vomiting and diarrhea and these usually lead to death in one to three days, so that a suspicion of poisoning is frequently created.

Treatment is unpromising, according to past experiences.

18. Inflammation of the Stomach and Intestines. Gastroenteritis.

Septic, typhoid, mycotic enteritis, intestinal mycosis, intestinal typhoid, dysentery of adult animals, Magenruhrseuche [German], *Fungus and meat-poisoning, croupous or diphtheritic enteritis, Mycosis seu sepsis intestinalis, gastro-entérite, gastro-entérite dysentérique* [French], *pseudo-typhoid*).

Under the name of gastro-enteritis are comprised all of those inflammatory processes which affect either the stomach or the intestines, but usually both of them simultaneously, and which are quite intense in character, leading to hemorrhage, suppuration or to the formation of true croupous membranes due to necrosis. These conditions do not constitute one specific entity,

but they have to be treated collectively under a common head since the necessary investigations are lacking to separate then properly into several specific types.

Etiology. In some cases **primary gastro-enteritis** develops after a simple catarrh of the stomach or intestines or it is brought about by the same factors which cause the latter. Lüdecke saw a hemorrhagic enteritis in cattle after the feeding of green sugar beet leaves which contained sodium nitrate. Similar cases have been observed in horses which had died in collapse before diarrhea had set in. Oemler observed many cases of the disease in two flocks of wethers after they had been pastured for a day on rice stubble with abundant aftergrowth, with the manifestation of hemorrhagic gastroenteritis (including the fore-stomachs) with diarrhea and intense reddening of the mucous membrane of the mouth (mortality 13.4 and 59.4%).

According to Ellenberger, enteritis, even hemorrhagic enteritis, is frequently observed after feeding rice shorts. Crushed rye containing castor beans may have a similar action. In Nevermann's case a hemorrhagic enteritis was produced by a mixture of linseed and bran. Bran has frequently had a noxious effect and has been the cause of many cases of sickness (see page 171).

With the exception of these types the most frequent causes of gastro-enteritis are spoiled feed (so-called **mould poisoning, gastro-enteritis mycotica, mycosis intestinalis**).

Herbivora sicken frequently after the ingestion of large amounts of spoiled and mouldy feed. As such may be mentioned rotten and fermenting beets or cuttings of beets, potatoes, to a lesser extent mouldy, moist grain or rough feed, particularly if much contaminated with rust-fungi, while Uredineæ and Ustilaginales in animal experiments have shown themselves to be harmless (Pusch, Von Tubeuf, Appel and Koske, Scheunert and Loetsch, Zwick, Fischer and Winkler), notwithstanding the fact that in the experiments of Liskun and Krassawizky on guinea pigs, mice, rabbits and in a dog these fungi were, upon autopsy, found to have had accumulated in all organs and were especially abundant in the fat of the kidney capsule and here and there had produced rupture of small blood vessels. Weber, however, observed some cases in a herd of breeding hogs following the feeding of crushed barley and barley shorts which were badly contaminated with Uredineæ and Ustilaginales. Molds form a heat resisting, exciting, tetanic and paralytic neurotoxin (Ceni and Besta, Baudin and Gautier), especially in spring and summer. In the observations of Heidrich a feed which was excessively contaminated with mites did not produce digestive disturbances in a horse. The ingestion of bedding straw and manure is sometimes also dan-

gerous. Water from wells in the neighborhood of cesspools, or water contaminated with animal offal has repeatedly caused numerous affections.

Carnivora very frequently develop a gastro-enteritis (meat poisoning) after the ingestion of infected or spoiled meat. Of first importance in this respect are the septicemias and pyemias which are caused by pus cocci (Puerperal fever, the several forms of wound-fever, purulent arthritis, purulent inflammation of the serous membranes, streptococcus mastitis, enteritis, etc.). The causative viruses of meat poisoning may also be of importance, however, and invading the meat or meat preparations coming from healthy animals may multiply in them (see meat poisoning). At times the meat becomes injurious through the action of specific viruses of septicemia (for instance in anthrax).

In 1895 numerous fatal cases of gastro-enteritis were observed, in the southern parts of Hungary, in dogs which had fed upon the meat of swine sick with hog cholera. It could not be decided in these cases whether the affection of the dogs was due to the microorganisms of hog cholera, to its toxins or to other bacteria. Berger saw hemorrhagic gastro-enteritis in the dogs of a village after these animals had fed upon parts of chickens that had been sick with fowl cholera.

The putrefactive spoiling of meat does not seem to be particularly harmful (M. Mueller), probably in part because toxic products are formed only if putrefaction is well advanced.

Otto observed a poisonous effect from salted mesentery in a dog. Glage has shown by animal experiments that meat meal produced by Podewil's method from putrid meat containing pathogenic microorganisms is not injurious. On the other hand, anthrax organisms are frequently found in fish meal, and to this the increased occurrence of anthrax in hogs must probably be attributed (Miessner and Berge). Fish meal also often contains, according to Miessner and Lange, short capsule-forming bacilli **(Diplococcus capsulatus)** pathogenic for animals.

Putrefaction of albumen leads to the formation of poisonous substances which may undoubtedly occasionally lead to meat poisoning characterized by paralysis of nerves. The most important of these are:

Neurin, a strong poison formed in putrefying meat after 5 or 6 days, probably in consequence of bacterial activity. Symptoms of neurin-intoxication are difficulty in respiration, weak heart, salivation, increased peristalsis, diarrhea, convulsions, finally collapse.

Muscarin is identical with the poison contained in the poisonous fly toadstool; it leads to similar symptoms.

Methylguanidin, another not well determined ptomain, is formed in putrefying horse flesh; it causes convulsions and paralysis of the heart.

Less poisonous are cadaverin and putrescin which cause local inflammations and death only in larger doses, cholin (poisonous only in very large doses) and finally a ptomain isolated by Garcia from putrefying horse flesh.

Pathogenic bacteria differing from those already mentioned and generally not yet well known may get into the intestinal tract with feed that is otherwise perfectly unobjectionable. Such bacteria probably cause dysentery of cattle, which sometimes

occurs enzootically among barn fed cattle, rarely on the pasture. The disease attacks almost without exception adult animals. There is also the possibility that inflammations may be caused by the normal intestinal bacteria in animals debilitated by insufficient feed. It is probable, on the other hand, that dysentery in adult cattle and sheep not due to coccidia is frequently in fact a form of hemorrhagic septicemia (see Vol. I). Whether the various intestinal inflammations which are designated as dysentery in animals, stand in any relation whatever to the bacillary dysentery of man, cannot be determined.

Gastro-intestinal inflammation in animals is often produced by intestinal bacteria which are present constantly or temporarily, and which may acquire a pathogenic action under certain circumstances which are not known definitely; this occurs probably after errors in diet, under unhygienic conditions, if the animals are underfed or overworked; under such circumstances these bacteria may become virulent and then become able to infect other animals without like contributing causes. Intestinal bacteria of this sort, which have become pathogenic in the animal body or, under favorable conditions, in the outer world, may live in the intestines as harmless saprophytes and may remain there after the enteritis due to them has passed off (bacillus carriers); consequently they may infect other animals.

Piorkowski and Jess have observed an enzootic enteritis on two large farms, which was due to a bacillus coli, and which led to perforation of the intestinal wall. Mohler and Buckley observed an epizootic among cows of one barn and were able to isolate a bacillus of the enteritidis group. Miessner & Kohlstock studied an enzootic of croupous enteritis in cattle on pasture, which had been caused by enteritis bacilli; the latter had been transmitted by a calf ill with persistent diarrhea, coming from a herd infected with dysentery. Similarly to the bacillus of enteritis, the paratyphoid bacillus B. may exert such an action. Kitt observed in a calf, morbid changes of the intestines similar to those found in hog cholera, namely band-like prominent diphtheritic pseudomembranes, concentrically arranged and button-like formations. The paratyphoid affections which occasionally occur in man after eating meat of animals slaughtered on account of sickness, permit the conclusion that intestinal inflammations, which may be hemorrhagic, are produced also in other domestic animals by paratyphoid bacilli. It is not impossible that the hemorrhagic enteritis of dogs, and also dog typhoid are due to like bacteria (see Vol. I). The etiological importance of the spirochetæ found in a case of gastroenteritis, by Lucet, is not established, because spirochetæ are common inhabitants of stomach and intestines.

According to Carré, the pseudotubercle bacillus of Preisz-Nocard causes a hemorrhagic and necrotizing colitis in horses, in which the anatomical changes correspond with those found

n hemorrhagic infarcts through obstruction of the mesenteric ırtery, and which could be produced artificially in two horses ›y pnre cultures of the bacillus. In accordance with this result, Carré includes all cases of "intestinal congestion" of the French veterinarians with the disease under discussion. A similar enteritis is met with in the course of the "Mal rouge" of sheep, which is due to the same bacillus.

Chaussé saw a peculiar form of hemorrhagic gastro-enteritis affecting everal animals in a large herd. It showed the usual symptoms of 'astro-enteritis, bloody feces mixed with blood coagula and an anemic iscoloration of the visible mucous membranes. Except in one case, all f the animals died, either after two to five days, or more frequently fter one to two weeks, after there had been some apparent improvement.

Post-mortem examination showed the changes of hemorrhagic eneritis with intestinal ulcers, and profuse hemorrhages into the intestinal 'all, extending into the peritoneum. The intestinal loops were matted ogether by blood. The mesentery and the pararenal adipose tissue howed gelatinous infiltration and the kidneys sometimes large hemaomata.

Intestinal bacteria may occasionally become pathogenic for nknown reasons. In the large intestines of the hog a croupous iphtheritic inflammation in the form of irregular patches is ound independently of hog cholera, which is due to the bacillus ecrophorus. Kitt saw a similar diphtheritic inflammation in he small intestines of horses, which was caused likewise by the ame bacillus. Bang and Berg found the identical microorganism as the canse of an enzootic enteritis of calves. If gastro-ıtestinal catarrh comes on after excessive efforts (long rides, articularly during great heat, long lasting railroad transportation) one may assume that a lowering of the resistance of he organism as a whole had led to a lowering of the resistance f the intestinal wall and had exposed it to the detrimental ffect of intestinal bacteria. The intestinal affection coming on within twenty-four hours after excessive work ("Distanzritt-rankheit" [German] of Heuss) has generally a hemorrhagic haracter and usually ends fatally. Graham, Reynolds and Hill vere able to transmit experimentally to mules the paratyphoid ue to Gaertner's bacillus in horses and mules following railroad ransportation (see Vol. I), only after the resistance of the nimals had been reduced by starvation and exhaustion.

Larger masses of sand which have found their way into the astro-intestinal canal sometimes cause gastro-enteritis in orses (Mazulewitsch, Wenderniikow and others) and in hogs, authors' observation) which often become affected when they re transported in railway cars the floor of which has been overed with sand. Herbivora may also receive food greatly dulterated with sand, in order to increase their apparent body weight (Haupt). In these cases also, intestinal bacteria prob-

ably are concerned, which are enabled to invade the intestinal mucosa partly on account of the mechanical irritation of the epithelial cells, partly because of the many erosions of the mucous membrane induced by the sand grains.

In the cases observed by Schnuerer in pigeons, especially in young birds, a diphtheroid enteritis with emaciation and diarrhea as a result of mechanical irritation of the intestinal mucosa by unsoaked oats (oat chaff) followed by coli infection terminated fatally in 5 to 8 days.

Secondary gastroenteritis sometimes follows after other affections of stomach and intestines and develops particularly if foreign bodies or parasites are present, or in case the contents of stomach and intestines accumulate in acute dilatation of the stomach, in obstipation, in obstruction of the intestine, intestinal stenosis and after non-fatal malpositions of the gut, also after a protracted hemorrhagic infarct of the wall of the intestine through thrombosis of the mesenteric artery. In these cases the mechanical injury to the epithelial cells, the free disintegration of the contents and the excessive multiplication of the intestinal bacteria afford a sufficient explanation for the relatively frequent occurrence of a deep inflammation in addition to these digestive diseases. Local and general infectious diseases may likewise be followed by enteritis, particularly in horses with severe pharyngitis (authors' observation), purpura and croupous pneumonia (Keilgaard, author's observation); in these cases the irritation may be of importance, being induced by bacterial toxins that are eliminated through the intestinal mucosa or are swallowed, and also the advent of pathogenic bacteria themselves into the gut, in swallowing or because of their elimination through the intestinal walls.

Anaphylactic enteritis also belongs in this catagory (see page 172). Finally gastro-enteritis develops frequently as the exclusive or partial localization of specific infectious diseases, of which are to be enumerated more particularly, anthrax, hemorrhagic septicemia, swine plague, rinderpest, influenza of horses, distemper of dogs, typhoid fever of dogs, etc. In horses, intestinal tuberculosis runs its course under the clinical picture of a protracted enteritis. The Nairobi disease of sheep and goats in British East Africa, due to a filterable virus and transmitted by ticks, also shows an acute febrile gastroenteritis (Knuth and du Toit). Certain intoxications commonly give rise to more or less severe gastro-enteritis, as poisoning with caustic or acrid substances (acids, caustics, phosphorus, arsenic, lead, mercury, carbolic acid, croton oil, cantharides, lupinæ, and other plants which contain acrid substances). Those forms of gastro-enteritis which make their appearance as sequels of specific infectious diseases and of poisoning, are not discussed in detail in this place, the reader being referred to the related

chapters in the first volume and to the textbooks on toxicology.

Pathogenesis. In comparison with the simple acute intestinal catarrh (see page 354), the development of gastro-enteritis differs only in so far as the injuries exert a stronger action and therefore produce not only degeneration but also the destruction of the intestinal epithelia. In this manner the substances, which give rise to the inflammation, invade the deeper layers of the mucous membrane, the submucosa and even the adjacent layers of the intestinal wall, giving rise to the formation of a fluid albuminous exudate, to copious secretion of mucus, migration of leucocytes and, not infrequently, to the extravasation of red blood cells. A rapid absorption of the toxic products takes place at the same time.

The formation of a copious, decomposable exudate in the lumen of the intestines induces a very rapid decomposition of the intestinal contents, with the production of irritating and poisonous substances, in which the superabundant multiplication of intestinal bacteria or of microorganisms taken up from the outside probably play an important rôle.

The formation of a copious exudate in the lumen of the gut, which is subject to disintegration, makes possible a rapid disintegration of the intestinal contents with the formation of irritating and toxic split products; in this process the excessive multiplication of intestinal bacteria, or of microorganisms originating from the outer world may contribute largely. With respect to poisoning by hyphomycetes, Tetzner assumes as probable the *formation of toxic substances owing to incomplete* cleavage of protein substances because the putrefactive bacteria *of the intestine* had been *influenced* by the *immense numbers* of hyphomycetes. The disintegration of the intestinal contents is promoted by the diminution or entire cessation of absorption. The intestinal motions are affected variously according to the composition and quantity of the intestinal contents and to the extent of the inflammation. In most cases they are accelerated because the substances which promote peristalsis gain the upper hand and neither the automatic nerve plexuses in the intestinal wall are injured nor is an inhibiting reflex action induced. Only if products of disintegration, which promote peristalsis, are lacking in the initial stage or if products are formed which inhibit the intestinal motions, is the peristalsis rendered slower; this may occur also if the intestine is empty, if the nerve plexus *of the intestine is involved or if reflex action through the sym*pathetic nerve is caused by severe and deep-seated inflammation. In a manner similar to the peristaltic motions, the frequency of defecation is affected, with the difference that efforts at defecation, may be induced independently of the peristaltic motions, by reflex action, if the distal portions of the colon or rectum are involved in the inflammation.

The general effects are usually severe. The organism is debilitated in consequence of the copious exudate, the marked loss of water and the severe circulatory disturbances, also because of thickening of the blood, through possible severe losses of blood, congestion of the region of the splanchnic nerve, absorption of toxic split products, of bacterial endotoxins and of bacteria. There is ample opportunity for the absorption of bacteria and of toxic substances because the epithelia of the gastric and intestinal mucosa are greatly degenerated or even dead, the digestion is arrested and the inflammation extends into the submucosa which is rich in lymph vessels. Toxic substances reach the digestive tract, in part as such, through eating spoiled and mouldy feed which may also contain infectious germs; they are then absorbed because they cannot be destroyed by the intestinal epithelial cells or by the digestive enzymes, or because they are present in very great quantities. The absorption of the bacterial endotoxins and of the products of protein cleavage give rise to fever and to injuries affecting the parenchymatous organs; the bacteria which enter the general circulation without being dissolved immediately, may produce local inflammation in distant organs or even a general infection which may gain the upper hand, especially in very acute cases of paratyphoid enteritis, as contrasted to the frequently insignificant local changes. An involvement of the peritoneum may be accepted as a sequel of the passage of microorganisms through the intestinal wall.

Anatomical Changes. The contents of stomach and intestines are often bloody and have a foul odor. The gastro-intestinal mucosa is affected to a variable extent from case to case, sometimes more uniformly, sometimes in patches, especially on the thick rugæ which are of a lively red color, either light or dark with punctate or streaked hemorrhages; there may also be more extensive suffusions of blood (gastro-enteritis hemorrhagica). In such cases the gastric mucosa in the territory of the small hemorrhages shows small roundish, sharply circumscribed superficial losses of substance (erosiones hæmorrhagicæ), the epithelial layer of the intestine may assume the appearance of a deposit like hoar-frost, ashes or bran, owing to the necrosis. In other cases the submucosa is swollen, edematous, again the stomach may contain purulent exudate (gastritis purulenta s. phlegmonosa). The hyperemic or hemorrhagic mucous membrane may be covered by dirty-gray or dull-yellow pseudomembranous deposits which either are quite superficial (gastro-enteritis fibrinosa s. crouposa) or have involved the deep layers of the mucosa (gastro-enteritis diphtheroides) and are then either diffuse or in spots, being found most frequently in the colon. After the necrotic portions of the mucous membrane have been exfoliated losses of substance or ulcers of variable size and shape remain, sometimes having

the appearance of a map (gastro-enteritis ulcerosa), and these may originate after the action of caustic substances without a prior formation of pseudomembranes. If the disease is prolonged, and especially in the presence of the bacillus of necrosis, the intestinal wall becomes rigid and thickened. The solitary and papillary follicles of the intestinal mucous membrane are always more or less swollen. The peritoneal covering of the stomach and intestine presents evidences of arterial injection, sometimes even inflammation. The mesenteric lymph nodes are acutely swollen. Frequent accidental findings are acute splenitis, degeneration or inflammation of the parenchymatous organs, rarely metastatic abscesses. (For the anatomical changes in paratyphoid of hogs, see Vol. I).

The gastro-enteritis due to the pseudotubercle bacillus of Preisz-Nocard is characterized, according to Carré, by a deep-red discoloration, necrosis and black-red softening of the mucous membrane in the colon, frequently also in the stomach; the heart muscle is yellow and friable; beneath the endocardium there are numerous ecchymoses, and on the valves fibrinous deposits. The liver has a cooked appearance and the cut surface of the spleen is black.

On post-mortem Stroh found lesions closely resembling human appendicitis in two rabbits that had died of disseminated paratuberculosis. Bélil-Bétat also applied the term appendicitis to an inflammation of the cecum of a mule which showed the greatest changes in a diverticulum of the cecum about the size of a thumb.

Symptoms. Gastro-enteritis sometimes begins with the symptoms of acute gastric catarrh and gradually passes over into the picture of inflammation; generally, however, the animals sicken suddenly under grave symptoms. The ingestion of feed and, in ruminants, rumination cease, thirst is sometimes much increased, often the animals exhibit manifestations of abdominal pains, which are generally dull, less frequently sharp and severe so that they lead to maniacal attacks. Carnivora and hogs vomit frequently, and the expelled matter occasionally is bloody; vomiting is sometimes seen in herbivora, and more frequently repeated retching and gagging.

All animals except the horse usually show tenderness on pressure of the region of the stomach or abomasum or even of the whole abdomen. In ruminants there is generally also moderate bloating with moderate increase of the circumference of the abdomen; this is, however, also in other animals, seen in the beginning of the disease. In the further course of the disease the dimensions of the abdomen usually decrease in consequence of diarrhea, and the abdomen is drawn in.

Whenever the stomach is affected particularly, the intestinal sounds are less frequent or entirely absent and there is constipation. If enteritis is already present the intestinal sounds are intensified or continuous and diarrhea sets in, the animals void

large masses of fetid matter with painful efforts; these are sometimes mixed with shreds of tissue or blood; dogs sometimes void pure blood; relaxation of the rectal sphincter muscle finally leads to involuntary defecation. If diarrhea has existed for some time and if the intestinal contents have consequently been diminished materially, the intestinal sounds, depending both upon peristalsis and the presence of fluids and gases, may be absent; simultaneously with the absence of intestinal sounds there occurs a decrease of the droppings and there may be complete constipation. However, tenesmus of the rectum is even then often observed.

In hemorrhagic gastro-enteritis of dogs loss of substance is seen frequently in the mucosa of the cheeks and on the gums, but this is rarely the case in dysentery of cattle.

The urine contains albumen, also occasionally formed elements such as casts, sometimes also blood, and it shows an acid reaction even in herbivora.

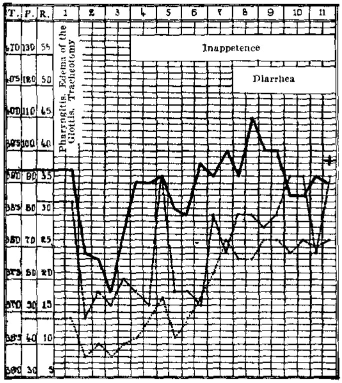


Fig. 12. Fever curve in diphtheritic enteritis in a horse.

The symptoms described are accompanied by great prostra-ion and grave general symptoms. An elevated temperature Fig. 12) sets in from the start, but the height of the fever aries considerably in different cases. In hemorrhagic gastro-nteritis of dogs the temperature soon goes down or it may be abnormal from the start. The pulse is accelerated from the eginning and it becomes small and soft. The heat distribution n the periphery is unequal, the extremities feel cold, the skin covered with perspiration and later on the eyes sink in.

Finally the animals lie on the floor stupefied and motionless, or they show convulsive motions, dizziness, muscular contractions, or their behavior is like that of dumb staggers. Hemorrhages into the skin, into the mucosæ or into the retina are sometimes observed (Schindelka).

Wirth found in hemorrhagic gastroenteritis in dogs an increase in the number of red blood corpuscles (up to 11,360,000) and of the hemoglobin content (to 140°) corresponding to the loss of fluid from the blood; also leucocytosis (up to 48,000 per cubic millimeter of blood) with distinct neutrophilia, increase in number of blood platelets and sometimes an increase in the coagulability of the blood. Sustmann observed pustulous exenthema between the toes and on the feet of tigers suffering from meat poisoning.

Croupous diphtheritic enteritis in the horse occurs in three different types. The most frequent type begins with great dullness and weakness of the patient, the gait is markedly staggering, the temperature rises to 40-41° C. or even higher, the conjunctivæ appear icteric, dirty discolored, injected. There is a lack of appetite, diarrhea sets in only after four to five days (see Fig. 45), the feces are at first mashy, then entirely fluid and fetid; they may contain pieces of pseudomembranes; before diarrhea has set in, dry feces may in rare cases be covered with shreds of croupous membranes. After diarrhea has appeared, a fatal issue generally soon occurs. The second type, according to Keilgaard, generally commences with not very severe symptoms of diarrhea; elevation of temperature may be absent and a moderate elevation of temperature may be noted only on the last day of the disease. However, the pulse is accelerated from the start. The general condition of the animals is greatly disturbed and a marked emaciation soon occurs. During the slower course of the disease, pseudomembranes are shed more frequently than in the first type. The third type, according to Folger, commences with the symptoms of sleepy staggers; the dull animals show a staggering, waddling gait; they lean forward, press against the wall, but do not show any symptoms on the part of the intestines. The disease usually ends fatally.

According to Froehner, poisoning by fungus is manifested as follows:

(1.) Hyphomycetes: Want of appetite, colic, tympanitis, constipation, diarrhea, bloody, mucous feces which sometimes have a foul odor, polyuria, vertigo, stupor, confused motions, paralysis of the extremities, of the tongue, etc., amaurosis, profuse diaphoresis. Tetzner observed a gradual development of enteritis with accumulation of feces in the colon, in consequence of paralysis of the cecum and colon (Friedrich held this so-called Senne disease [so called from the military drill grounds, Senne] to be Borna disease); Berndt, besides these, noted symptoms of paralysis. Mayer observed the following phenomena: 1, Severe colic accompanied by excitement, with recovery in 2 to 5 days; 2, contrasted with the above, the colic would terminate sooner and be followed by gastroenteritis lasting for 3 to 4 weeks, beginning with constipation and followed by a fetid and sometimes bloody diarrhea; 3, a clinical picture resembling lumbago, light colic, and slight depression of the sensorium, soon followed by paralysis of the hind quarters; 4, icterus lasting several days.

(2.) *Ustilaginae;* Salivation, persistent motions of mastication, swaying, reeling, general motor and sensory paralysis; in other cases, gastritis.

(3.) Uredineae: Dermatitis on the head (lips, cheeks, eyelids) conjunctivitis, urticaria, stomatitis, pharyngitis, glossitis, colic, bloody defecation, hematuria, paralyses, somnolence.

(4.) Saccharomyces: Severe cerebral excitement with subsequent stupor and symptoms of paralysis.

An enteritis observed in dogs by Bergeon, after feeding with rice, which this author looked upon as a beri-beri like affection, produces rapid exhaustion and dyspnea with violent action of the heart; later, diarrhea with the discharge of black, ill-smelling fecal matter, also marked thirst and diminution of the appetite. The posterior extremities are frequently weak as though paralyzed, there may be necrosis of the skin, ulcer of the cornea, rarely albuminuria, but no particular rise in temperature. Death occurred after 20-30 days. The disease was prevented by giving meat.

Course and Prognosis. In very grave cases the disease usually terminates fatally within the first two days, even within the first twenty-four hours, while in ordinary cases it lasts from one to two weeks before death comes on from exhaustion, or a fatal issue may also occur after an apparent improvement. Recoveries are as a rule rare and convalescence is of very long duration.

The more rapidly the symptoms become worse, the less chance is there for recovery. Important unfavorable signs are a considerable acceleration of the pulse, profuse and bloody diarrhea or obstinate constipation. There are, however, cases, particularly among herbivora with a long intestinal canal, where there may be a mild course of gastro-intestinal catarrh extending over one to two weeks, which may suddenly become worse and end in death in one or two days. Enteritis in hogs, due to the bacillus necrophorus, not uncommonly takes a less virulent course.

Diagnosis. A correct diagnosis is usually made possible by the sudden onset and the rapid aggravation of the disease with the simultaneous occurrence of abdominal pain, also the fact that, in herbivora, obstinate diarrhea is either present from the beginning or follows upon constipation; the great debility sometimes associated with severe nervous disturbances, the decided acceleration and weakness of the pulse are characteristic. In some cases it is possible to determine the cause of the primary affection from the case history, the examination of food, pasture and drinking water. The symptoms just enumerated differentiate gastro-enteritis from simple gastro-intestinal catarrh in the course of which general disturbances usually are absent or only slight, and in which appropriate treatment rapidly leads to improvement or cure. Secondary gastro-enteritis arising from other affections of stomach or intestines (dilatation of the stomach, fecal impaction, intestinal concretions, helminthiasis,

etc.) is preceded by the symptoms of the respective diseases, and the association of gastro-enteritis is announced by the appearance of fever and by marked acceleration of the pulse. In parasitic affections of stomach and intestines, the macroscopic and still more the microscopical examination of the intestinal discharges give decisive information. It is important to eliminate secondary gastritis in certain acute infectious diseases, especially in anthrax, hemorrhagic septicemia, swine plague and rinderpest. Aside from the examination of the food, it is important to inquire into the mode of development of the disease, also into its sporadic or crowded occurrence, and particularly into the changes in and on the skin, which are characteristic of these diseases, also in the buccal cavity, in the respiratory organs, and in the eyes; further, the frequent absence of colicky pains. Intestinal tuberculosis and paratuberculous enteritis are recognized with certainty only by the aid of microscopical examination of the feces or of scrapings of the mucosa from the distal portions of the colon; intestinal tuberculosis in horses may be diagnosed through rectal examination of the mesenteric lymph nodes and of the spleen. In horses and cattle the possible existence of these diseases should be considered if the enteritis takes a very slow course. In order to exclude poisoning, the composition and condition of the feed is of importance, also of the plants on the pasture, and of the drinking water; further, the history of the case with reference to treatment of the animals with sharp and caustic remedies and to the disinfection of the barns with such substances, also the possible simultaneous occurrence of the same symptoms in several animals, the afebrile course of severe intestinal disturbances, and the pathological symptoms in other organs, more particularly in mouth and pharynx, in the air passages, eyes and on the skin.

Treatment. If the sick animals are still in a fair state of nutrition, they should be starved several days; the tormenting thirst must, however, be alleviated by lukewarm water. Dogs should also receive tea with some cognac. If there is great debility mucilaginous food should be given, such as soups of linseed, oatmeal, rice, sago with the addition of alcohol and eggs or wine, soups with the yellow of eggs, if the condition of the stomach permits feeding by mouth at all. If this is impossible, artificial feeding must be instituted (see page 43); this may be accomplished by rectal feeding, or if the rectum is likewise affected, by subcutaneous or perhaps by intravenous injections. If gastro-enteritis is due to the ingestion of spoiled feed, the early removal of the gastro-intestinal contents is the first indication. Emetics by the mouth or by subcutaneous injection may be used in hogs and in carnivora; but much better *is lavage of the stomach (see page 131); in other animals efforts* must be confined to the administration of mild laxatives such as castor oil, salts, calomel (see page 144). Subcutaneous injec-

tions of drugs stimulating peristalsis, (eserine, arecoline, etc.) are less advisable, although they may be indicated in such cases where abdominal pains are absent. If there is reason to accuse noxious microorganisms introduced with the feed as the cause of the disturbance, one should attempt to prevent their multiplication in the intestinal tract by the administration of disinfectants such as resorcin, naphthalin, creolin, salicylic acid. Raebiger suggested medicinal methylene blue Hoechst (mature cattle 5 to 10 gm. on feed, in capsules or pills; calves and sheep 0.5 to 1 gm. in water or milk). Homblau may also be tried. Mucilaginous drugs and astringents (see page 182), and especially animal charcoal (see page 183) and incarbon (horses 120 cc. repeatedly), can be used to advantage. Protein therapy may also be attempted (see croupous pneumonia) by repeated subcutaneous or intravenous injections of serum foreign to the body (dogs and pigs 1 to 2 cc., calves and hogs 20 to 30 cc., horses and cattle 100 to 150 cc.), or by the subcutaneous injection of milk sterilized by boiling (same quantity), with which Dasch claims to have obtained astonishingly favorable results in hemorrhagic enteritis in dogs (20 cc.), while such a therapy was without the slightest effect in 1,000 cases of dog distemper. In the hemorrhagic forms the following drugs may be used as hemostatics: Adrenalin, tonogen, paranephrin, suprarenin in 0.1% solution (0.02 to 0.04 cc. of the 0.1% solution per kg. body weight subcutaneously, 0.01 cc. intravenously; for dogs internally 30 drops every 3 hours, or in all 0.2 to 0.5 cc. subcutaneously); also Witte's peptone in 5% solution with physiological serum (after neutralization with sodium carbonate, dogs 15 gm. internally daily, after boiling and filtering, 10 to 20 cc. subcutaneously). In cases of poisoning, antidotes, lavage of the stomach and mild laxatives are indicated.

If abdominal pains are severe they must be alleviated by opium or morphine (see page 145): prostration and stupor should be treated by cold douches and friction; however, douches must not be employed if the temperature is subnormal, when warm packs are indicated. A weak pulse calls for the administration of stimulants such as wine, whiskey, black coffee, tea internally; ether, oil of camphor, caffeine subcutaneously—in weakness and collapse. The infusion of warm physiologic salt solution into a vein or under the skin may be very beneficial, particularly if some adrenalin, suprarenin, or 4% grape sugar solution has been added; such infusions may be repeated several times.

Enteritis in Fowls. Aside from secondary enteritis which occurs in the course of fowl cholera, hemorrhagic septicemia of chickens and other fowl-septicemias, in fowl diphtheria, in poisoning by acrid substances (in pigeons also after eating seed in the field which had been treated to prevent smut), and finally in the presence of intestinal parasites (coccidia-enteritis, helminthiasis), primary croupous-diphtheritic

processes are met with in the intestines of chickens, geese, ducks, turkeys and peacocks (Röll, Johne, Kitt, Guittard). Such affections may appear sporadically in animals of one species only or in animals of several species.

The **cause** of croupous-diphtheritic enteritis in fowl is not well known; however there is no doubt that one must look for infectious agents as their cause.

• Fumagalli has seen an enzootic croupous enteritis in chickens caused by aspergillus.

Post-mortem examination shows, either in the small intestines (Röll, Kitt) or only in the cecum (Guittard), soft cylindrical croupous-diphtheritic masses as long as a finger, filling the intestines more or less completely; the mucosa is intensely reddened, swollen and hemorrhagic. In other cases the intestines contain a smeary-purulent, rice-waterlike or light reddish-gray discolored fluid; while the mucosa is either in a condition of serous infiltration and partial softening (Röll) or is covered by a veil of fibrin coagula, or it shows sharply contoured, blackish or greenish-gray places, elevated 3-4 mm. above the surface; still other evidences of inflammation may likewise be seen.

The **symptoms** of the disease are lack of appetite, depression, sluggish motion due to stiffness of the muscles, brown discoloration of the integument of the lower abdomen, violent diarrhea with yellowish droppings.

The disease usually takes a rapid course and the animals die often within a few hours (Röll) or after the disease has lasted from twenty-four to thirty-six hours; if the ceca alone are affected, death may follow in only seven to eight days.

The **treatment** is unpromising. Guittard recommends as a prophylactic the administration of a decoction of radix altheæ, and the addition of syrup, honey and naphthol to the feed; the latter should previously be softened in a weak solution of carbolic acid or salol.

Dysentery in Man. The term dysentery will be reserved for the diphtheroid and ulcerous enteritis caused by the dysentery bacillus and will be considered as distinct from amebic dysentery (see amebæ in the intestinal tract). Bacillary dysentery occurs epidemically in all climates and is especially prevalent during wars. It is caused by **Bacillus dysenteriæ** Shiga-Kruse, in other cases by the Flexner bacillus or Bacillus Y, and also by the bacilli of His, Strong and Saigen, the latter group of which are less toxic and provoke less severe symptoms than the Shiga-Kruse and Flexner or Y bacilli, because these latter, owing to the absorption of their toxin, cause severe nervous disturbances and frequently a fatal termination.

The anatomical changes in bacillary dysentery consist in catarrhal inflammation of the mucous membrane with subsequent swelling of the lymph follicles, necrosis of the epithelial cells resulting in transverse ulcers, diphtheritic deposits in the colon.

The symptoms are those of gastro-enteritis with severe tenesmus; the fecal dejecta are at first thin-mushy, purely mucoid, feculent, later foul or having an odor like sperma; in course of time they are mixed with pus, blood and shreds of epithelium. Slight fever is usual, subnormal temperature infrequent; para- and hemiplegia occurs, also paralysis of individual nerves, nervous cardiac symptoms, inflammations of joints

and tendon sheaths. The mortality is as high as 10-20% and even 35-50%. Sometimes a chronic dysentery persists with slight diarrhea.

True Dysentery in Animals. While, according to Lentz, Bowmann and Werner, bacillary dysentery is said to be peculiar to man and otherwise found at the most only in laboratory monkeys, a disease very similar to dysentery in man can be produced in rabbits by the intravenous injection of dysentery bacilli. Dold even observed primary bacillary dysentery in dogs in valleys in China where the hunting grounds of the diseased dogs had been abundantly fertilized with human feces. All 3 of the above first-named species occurred separately in individual cases, the serum of the patient agglutinating them in a dilution of 1:300-100. A dog infected artificially per os, though he did not become sick, remained a carrier for 3 months.

The symptoms observed were bloody intestinal discharges mixed with mucus and accompanied by emaciation. One animal died after two weeks; the others recovered in the course of several weeks.

Meat poisoning and Botulism in Man. Meat poisoning usually occurs after the ingestion of meat from cattle, calves, cows, sometimes also hogs or even horses which were ill of septic or pyemic inflammatory diseases when they were slaughtered, such as puerperal fever, purulent mastitis, purulent inflammation of the serous membranes or of joints, enteritis. The apparently normal meat from animals ill with other diseases, or from healthy animals, may give rise to meat poisoning if intestinal bacteria have invaded the circulation or if the meat has been contaminated through being handled by persons who have pathogenic paratyphoid bacilli in their intestines although in paratyphoid enteritis the symptoms may not be very pronounced, so that the meat can not be recognized as dangerous for human consumption until it has been examined bacteriologically. Severe endemics occur particularly in summer.

Among the causes of meat poisoning various poisonous substances enter into consideration which are preformed in spoiled foodstuffs; usually bacteria are also responsible. Aside from pathogenic strepto- and staphylococci, the paratyphoid bacillus B. is of special importance. Exceptionally paratyphoid bacillus A, which closely resembles **Bacillus typhosus** and can be distinguished from it only by certain cultural characteristics and immunity reactions, plays a role. Bacteria of the colon group and the proteus bacillus are of little importance.

In many cases the symptoms correspond with those of a slight gastro-intestinal catarrh, in other cases they are those of febrile gastro-enteritis or even of a typhoidal disease with muscular weakness, ataxia, frequently with albuminuria, bronchopneumonia, circumscribed erythema of the skin, urticaria and cutaneous hemorrhages. In contrast to abdominal typhoid, the disease commences with fever that rises sharply and is atypical; the feces assume the appearance of pea-soup only perhaps in the later course of the disease; splenic tumor is usually absent. The diagnosis can be established with certainty by demonstrating the bacteria of meat poisoning, in the blood, urine, feces, or in the juice obtained from the roseolae, if these substances are planted in bile or bouillon and after 24 hours' incubation on litmus-milksugar-agar and on malachite-agar. In the later part of the disease, the agglutinin test is positive. In spite of the serious symptoms, the mortality is hardly more than 2-5%.

Sausage poisoning (botulism, allantiasis) has been noticed after the ingestion of sausage, corned or smoked beef, canned beef, conserved

meat, venison, etc. Sometimes similar symptoms have been observed after the ingestion of salted fish (ichthyosism).

According to the compilation made by Bitter, the following foods were responsible in 302 cases of botulism with a death rate of 16%: Hams and sausage 17 times; bacon and smoked meat, each 1 time; lobster and pickled fish 12 times; other meat dishes 9 times, and preserved beans 1 time. An unusual odor and taste of such food was often observed. The infection with **Bacillus botulinus** could be traced to insufficient cooking and smoking, too low acid content (less than 2% acetic acid), insufficient salting (less than 10% sodium chloride content of the solution used) and lack of cleanliness in preparation and storing.

The cause of sausage poisoning are the toxic products (botulism toxin) of the anaerobic bacillus botulinus of Van Ermengem, which is found in meat preparations and taken with them into the gastro-intestinal tract of man. This toxin produces cloudy swelling and fatty degeneration in the cells of the parenchymatous organs and the lining endothelia of the blood vessels, also changes in the ganglion cells of the anterior roots of the spinal cord and in the bulbar nuclei.

Hence we observe in botulism symmetric motor paralysis, particularly in the region of the cerebral nerves (paralysis of accommodation, mydriasis, ptosis, aphonia, dysphagia), while gastro-intestinal disturbances are not infrequently absent or are only very insignificant. There is, however, obstinate constipation and suppression of urine.

According to Semmerau and Noack, botulinus toxin can be readily detected in the blood of the patient by animal experiment. Mortality is from 25 to 30%.

Botulism in Animals. According to Dickson, repeated multiple cases of botulism were observed in America in chickens which had consumed preserved string beans which were also toxic for man. The chickens showed droopiness, laziness, lack of appetite, rapidly increasing weakness of the legs, wings and neck, dropping of the wings and supporting of the beak or one side of the head upon the ground; hence, they showed all the symptoms of so-called "limberneck" (a designation used for various other diseases in which the head assumes a similar position). Histologically peculiar thromboses and hemorrhages were found in the brain. In the observations reported by Hart 500 out of 643 chickens died with similar symptoms. Ducks and geese are also reported to be susceptible. (The symptoms of limberneck were produced artificially by Dutcher and Wilkins by feeding larvæ of **Lucilia cæsar** that were collected from limberneck cadavers.)

According to American veterinarians, botulism may also occur in horses after eating seemingly good oat straw. Such cases are characterized especially by sudden blindness. (Others traced cases with simultaneous excitement to the consumption of cotton-seed meal.) In swine botulism has been reported to have been transmitted by the use of hog-cholera serum that was contaminated with **Bacillus botulinus.** It was characterized by stiff and unsteady gait, weakness, sometimes paralysis of the posterior parts, accelerated or difficult breathing, occasionally with conjunctivitis with profuse mucopurulent discharge and usually also disturbances of vision.

19. Bloating of the Intestines. Meteorismus Intestinalis.

(*Darmaufblähung, Windkolik* [German]; *Colica flatulenta; Indigestion intestinale gazeuse* [French].)

Bloating of the intestines or meteorism consists in an excessive dilatation of the intestines in consequence of the rapid formation of gas.

Occurrence. The prevalence of primary meteorism is intimately connected with the methods and conditions of feeding the animals. These conditions vary a good deal in different countries and parts of countries, hence the affection is not uniformly prevalent everywhere. In the Budapest Clinic it represents 12 to 15% of the colicky affections of horses; the lower percentages, however, are observed only rarely. In the clinic at Copenhagen the percentage in the years 1892-1912 amounted to 6.4% (Brieg); in Vienna during 10 years to 7% (Wirth). Occasionally the affection occurs in hogs, dogs and rabbits. In ruminants the same causes lead to bloating of the rumen.

Etiology. **Primary bloating** of the intestines is due to the ingestion of bloating feed. Particularly dangerous in this respect are withered or heated fresh green feed (clover, alfalfa, esparsette, fresh grass), also beets and potatoes, rarely seeds of leguminosæ, more frequently crushed corn or barley. Mouldy feed may likewise be dangerous (Hendrick). Bloating in rabbits is not infrequently caused by fresh cabbage, kale, beets, rape, vetches, buckwheat. The ingestion of water in large amounts increases the danger from these foods materially. Sometimes an unobjectionable feed, like oats and hay may cause bloating in horses if these animals feed greedily and rapidly or are overworked shortly after feeding. Dogs are sometimes bloated after the abundant ingestion of starchy food. Abundant stuffing of parts of the intestines with thick mashy feces favors the collection of gases in the parts nearest to the stomach.

Secondary bloating occurs in various forms of closure of the intestines in grave thrombotic-embolic disease of the intestine; and in the course of diffuse acute peritonitis, in internal strangulation, volvulus and thrombosis of the mesenteric vessels, bloating appears early after a few hours in the affected loops of intestines, while anteriorly to the obstruction, as in other cases of secondary bloating, this condition develops slowly and only rarely reaches a high degree during the life of the animal.

Pathogenesis. A portion of the feed swallowed by horses gets from the stomach into the small intestines even during feeding and from there soon into the large gut. According to Scheuner and Grimmer corn gets into the large intestines two hours after ingestion, hence fermentable feed can form gases, very shortly after ingestion, along the whole intestinal tract with the exception of the rectum and also to a moderate extent in the stomach. In consequence of stretching and the chemical influences (carbon-dioxide, methane, fatty acids) the muscularis of the stomach and intestines is irritated to frequently recurring convulsive contractions which in their turn produce colicky pains. The strong contractions and the absorption of the gases by the blood at first prevent great

stretching of the intestines, but these means are insufficient to absorb all of the rapidly forming gas. Loops of intestines, therefore, become more and more dilated; these, as well as the whole intestinal tract, suffer in contractility while compression of blood vessels occurs likewise, further preventing the absorption of gases. The dilated intestines also press the diaphragm toward the thorax and the negative intrathoracic pressure is diminished, the cardiac diastole becomes interfered with and the blood pressure in the arteries is lowered.

In hogs and dogs bloating is developed similarly though generally somewhat later after food ingestion.

Anatomical Changes. On postmortem examination the abdomen is occasionally dilated and very tense, the more so since fermentation and the formation of gases continue after death. The dilated and tense loops of intestines are pressed out with great force after opening up the abdominal cavity. If rupture has occurred gastric and abdominal contents are found free in the abdominal cavity. The margins of the tear show bloody suggillation and possibly also swelling. These are anatomical signs of death by suffocation which are absent if gas formation is post mortal.

Symptoms. In **horses** symptoms of primary bloating usually appears shortly after the ingestion of food and are similar to those of acute dilatation of the stomach (see page 140). As a rule the clinical picture is initiated by violent, frequently recurring attacks of colic. Since genuine colicky pains, due to convulsive contraction of the intestinal muscularis are not intensified by pressure, the patients throw themselves recklessly on the floor and roll about in a manner attracting attention; later on they sit down on their haunches (dog position).

Simultaneously with the symptoms of colic, the abdominal circumference increases rapidly, the space between the costal arches becomes greater, the abdominal wall protrudes in barrel shape; most marked is the increase of the abdomen in the flanks, particularly on the right side. Animals that normally possess a tense and less yielding abdominal wall, will not show a very well marked enlargement of the abdomen, in spite of severe disease.

The percussion sounds are deep and resonant, more or less all over the entire abdomen, but particularly in the flanks; the sound becomes, however, somewhat higher and weaker in grave cases on account of the great tension of the intestines and of the abdominal wall. Sometimes the sound is metallic.

The intestinal sounds are sometimes continuous at the beginning of the affection; in the further course of grave cases they become less freqnent and may cease entirely; they are often high in pitch and metallic.

Rectal examination shows a high degree of dilatation of all

accessible intestines with the exception of the rectum. Their walls feel tense and elastic. Bloating of the colon displaces its pelvic flexure deep down into the pelvis or towards the right side and in the latter case the tense longitudinal bands of the left lower portion run from the right towards the left and sometimes in a spiral arrangement. The left lower portion is now much dilated and reaches up to the left kidney; it is either beside or below it and the much thinner, but likewise bloated left upper portion of the colon is displaced by the raised lower portion, either towards the median line or towards the left abdominal wall (Fig. 13).

Defecation at first occurs more frequently but soon becomes delayed or suppressed. In the beginning, and in the less severe cases until termination of the affection, there is a good deal of flatus.

The respiration becomes increasingly more difficult; the pulse rapidly becomes accelerated, so that when the bloating of the posterior abdomen has become quite noticeable the pulse will soon be 60 per minute; the more rapid it becomes the weaker it will be. The mucous membranes are at first dark red, soon, however, they become cyanotic, the visible veins are strongly filled.

The body of the patient is bathed in perspiration. If the stomach is likewise bloated there is also often belching, more rarely retching, occasionally vomiting.

Wöhner saw a case of bloating in a foal with subcutaneous emphysema on the back and shoulders. The case ended in recovery.

Secondary bloating leads to variable symptoms aside from the increase in abdominal circumference; these depend largely upon the nature of the underlying condition.

In **hogs, dogs** and **rabbits**, the clinical picture varies somewhat from that in the horse; the former animals either do not suffer abdominal pains or they only betray them by repeated attacks of crying out, frequent changes of position or groaning. Belching, retching and vomiting on the other hand are seen more frequently.

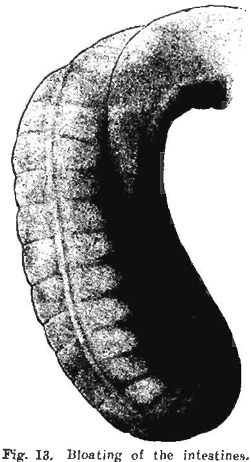


Fig. 13. Bloating of the intestines. Position of the left loops of the colon and course of the longitudinal bands of the left lower portion in extensive bloating of the horse.

Course. The accumulation of gas may reach such a degree, *occasionally within four to five hours, and in smaller animals earlier*, even if the animals have apparently quieted down, that suffocation comes on. (Goldbeck saw a horse, in which bloating came on after eating fresh clover, succumb after the illness had lasted only 16 to 20 minutes.) Ruptures are sometimes seen; they occur in those cases of primary bloating where the accumulation of gas is not uniform and where some portions of the gastro-intestinal tract become excessively dilated. (Rupture is more frequently seen in the stomach. The diaphragm tears occasionally.) (A blunt object acting with force upon the abdomen may rupture the normal intestine, stomach or rumen.) If rupture has occurred, the animals become suddenly quiet; however, the general condition deteriorates and collapse occurs rapidly (see page 141).

The affection not infrequently ends spontaneously in recovery; in the majority of cases recovery can, however, only be expected upon proper treatment. The course is always of short duration and usually it becomes obvious after twelve hours whether death will occur or whether improvement may be expected; if the latter is the case the symptoms of acute intestinal catarrh generally set in.

Diagnosis. The most important symptoms of primary intestinal meteorism are the following: rapidly increasing signs of colic after the ingestion of usually bloating feed, rapid increase in the size of the abdomen, loud sounds on percussion, and uniform, extensive dilatation and tension of all of the intestines (general intestinal meteorism). Simultaneously bloating of the stomach may be recognized by belching and vomiting.

Secondary bloating (see torsion or strangulation of the intestines; accumulation of feces, thrombosis of intestinal arteries, intestinal obturation, enteritis, peritonitis), which must be judged and treated differently, may be initiated with similar symptoms and must be excluded upon the basis of the history *and upon the result of a rectal examination which should be* made in every case of meteorism. Except in the case of peritonitis, secondary bloating is always confined to individual portions of the intestines (circumscribed, intestinal meteorism), and there are often symptoms which clearly point to the causative affection. Bloating occurring in consequence of an inflammatory condition is accompanied from the start by fever and the restlessness is not so marked. Torsion of the stomach in carnivora may be excluded when belching occurs and when the abdominal wall is not tender to pressure.

Treatment. In not too severe cases of primary meteorism one should attempt to stimulate intestinal contractions in order to remove the gases per vias naturales. Cold packs to the abdominal wall, cold douches to the latter, and cold water injected

under some pressure into the rectum, favor the expulsion of gases in the horse by reflex irritation. One may also employ ether in water (15:400), salt solution, soap suds or much diluted turpentine. Massage of the abdomen or internal massage of the colon and cecum, applied with care through the rectum, are also beneficial; however, if the intestines are very tense one must not use this method, because rupture might be caused by it. Hummerich produced an extensive evacuation of the intestines by rolling the previously restrained horse on the back 15 to 20 times and then after an interval of time 8 to 10 times; this procedure may have to be repeated three or four times. Laxatives are also administered (salts with aloe, 150-200 gm. to 15-20 gm.); disinfectants are added to these to reduce fermentation (lysol or creolin, 15-20.0 gm. naphthol, naphthalin, 10-15 gm.). As long as the contractility of the intestinal loops is not abolished, as long as evacuation by it has not become impossible, and as long as intestinal gases are expelled from time to time, eserine (0.08-0.10 gm.) or arecoline (0.06-0.08 gm.) may be used subcutaneously. If, however, parts of the intestine have already lost their contractility (in very severe cases), these drugs are no longer beneficial and may be dangerous (see page **144**). Since bloating of the stomach is frequently present simultaneously, the stomach tube should be used.

If, in spite of these applications, the condition of the patient becomes worse, or if the excessive dilatation of the abdomen bring about great dyspnea, puncture of the intestines must no longer be delayed. This removes the danger of suffocation and reestablishes contractility of the punctured portion of the intestines and the parts situated between the puncture and stomach. In secondary bloating intestinal puncture alone promises relief. Puncture of the cecum is usually practiced; if necessary, however, the colon or the small intestines must be punctured.

Intestinal puncture is best practiced with a slender trochar 10–15 cm. long. The skin is first properly cleansed, then an incision is made. The trochar is now introduced into the center of the depression of the right flank with the point directed towards the left elbow. After the escape of gas ceases, the tube is removed or, better still, left in place and closed with a cork, when it may be opened later on to let out more gas which might have again accumulated. If in spite of this puncture the symptoms of bloating persist or if the left flank has been distended more prominently from the start, the left flank is punctured. If this is done one must previously ascertain by rectal exploration at which point of the left side the bloated colon is situated, so that one does not puncture the rectum or some loops of small intestines or enter into the free abdominal cavity, in which case the puncture is without any result whatsoever.

Puncture of the colon from the rectum (Imminger, Föringer, Jensen, Reinhardt) appears indicated only in those rare cases, when the left portion of the colon is not lying close to the abdominal wall and when puncture of the cecum does not bring relief. Such a puncture may be made with a large exploratory trochar or an Elschner trochar with a curved canula 40 cm. long or a Raitsits trochar which is short and can be introduced into the rectum as far as one can reach, and may in this manner reach anterior portions of the intestines. To permit the escape of the gases the short trochar must be connected with a rubber tube. If the free end of the tube is placed below water the evacuation of gas can be easily controlled. When a proper trocar is not at hand the regular cecum trocar may be introduced into the rectum after a string has been attached to its stylet.

Intestinal puncture, whether practiced from the flanks or from the rectum, is never harmful in primary bloating, if we use a slender trochar and proceed under aseptic precautions, or at least as cleanly as possible, and if in puncture from the rectum one follows the trochar with the guard. Before puncture from the rectum, the latter should be irrigated several times with disinfectant solutions. In secondary bloating, when the elasticity of the intestinal wall has suffered in consequence of serous or hemorrhagic extravasation, it may occur exceptionally that some intestinal contents get into the abdominal cavity.

Great restlessness must be counteracted by morphine injections (0.3-0.5 gm.) or by rectal injections of chloral hydrate, because reckless rolling may bring about rupture.

In hogs, dogs and rabbits, kneading or massage of the abdominal cavity may be beneficial; also chasing the patients around, also repeated cold douches. If the stomach is bloated simultaneously, emetics (see page 131) may be used. If suffocation threatens, these animals likewise require intestinal puncture. The latter is practiced in the middle of the left or on the right side of the abdomen.

20. Accumulation of Feces in the Intestines. Impaction. Obstipatio.

(*Kotanschoppung im Darm, Verstopfung, Hartleibigkeit* [German]; *Indigestion intestinale par surcharge* [French].)

Obstipation or impaction consists in an accumulation and drying of masses of feces in the intestines with subsequent progressive enlargement of some portions of the intestinal tract, which condition may finally lead to a complete closure of the intestinal lumen.

The disease defined as above has a definite clinical and pathologic-anatomical picture and may be differentiated from other forms of fecal accumulations accompanying displacements, circumscribed paralysis, obturation of the gut, also acute peritonitis and acute diseases of the stomach.

(a) Obstipation in the Intestines of the Horse.

(*Anschoppungskolik* [German]; *Colica stercoracea; Indigestion intestinale par surcharge* [French].)

Occurrence. The frequency of primary obstipation is largely determined by the type of feed and hence the disease varies very much as to occurrence in various parts of the world. In the Budapest Clinic obstipation varies in different years from 6 to 20% of the cases of colic observed; in the Berlin Clinic Behrens observed in 1909 the unusual percentage of 74, and Frese in 1911-12 observed it in 94% of all colic cases, while at the Copenhagen Clinic Brieg in 1892-1912 observed this form in only 22.64% of all colic cases. In the years 1909-18 Wirth reported it in the Vienna Clinic in 46% of all colic cases, while

in 1915-16 it amounted to 40%, and in 1915-16 to 1918-19, when the feed was of poor quality, to 53%.

As the frequency of obstipation varies, so its localization to individual parts of the intestinal tract is variable. Of 1,225 cases of intestinal obstipation observed at the Budapest Clinic in the years 1906-19 the localization was as follows: Colon 851 cases, colon and cecum 81 cases, ileum 44 cases, duodenum 3 cases, cecum 157 cases, small colon and rectum 89 cases. At the Copenhagen Clinic in the years 1892-1912 in 717 horses with obstipation terminating fatally the distribution was as follows: Colon 388 cases, cecum 295 cases, and small intestines only 34 cases. At the Berlin Clinic in 542 cases of fatal obstipation in the years 1897-1911 the localization was found as follows: Colon 296 times, ileum 161 times, duodenum 41 times, rectum 14 times, and the jejunum 10 times. Behrens in 1909, out of 547 cases of obstipation of the intestines, found 316 to be due to obstipation of the small intestines and 231 to that of the large intestines, and gave the proportion as being 4:3. This is contrary to the general observation in other clinics, as is also the similar report by Frese on 326 cases of obstipation of the small intestines and 271 cases in the large intestines out of a total of 634 cases of colic in the years 1911-12. The observation of Reinhardt should be mentioned, according to which obstipation of the cecum and colon did not occur at all in the Berne Clinic (green feed as well as dry feed).

In the observations of Trippeers the obstipation, when straw was fed, was found to be distributed as follows: In 80% of the cases in the cecum, in 5% the transverse (diaphragmatic flexure) layers of the colon, in 10% the stomach-like enlargement, in 5% the small colon and the rectum. When barley was fed with the roughage the percentage of the transverse layers was increased and that of the cecum decreased. When alfalfa hay alone was fed obstipation was found in 85% in the small colon, in 10% in the stomachlike enlargement, in 3% the superior transverse layer and in only 3% in the cecum. According to the compilation of Wirth from the Vienna Clinic, the number of cases of obstipation of the small intestines increased from 0% to 25% in years when the feed was of very poor quality; in somewhat more favorable years the percentage of fatalities due to obstipation was 21.9%, and the number of cases of obstipation of the cecum decreased by more than two-thirds, as was the case also with catarrhal intestinal convulsions, while the number of cases of tympanitis and dislocation of the intestines remained about the same.

Etiology. The long continued ingestion of feed rich in cellulose and wood fibers frequently plays a rôle in the production of obstipation in horses, because feed of this type has to be taken in very much larger amounts than more nutritious food and it furnishes feces of much denser consistency, which are more difficult to move along. Such food stuffs are straw, particularly if cut into short chaff, or if it is taken up from the bedding straw (socalled straw feeders), corn stalks, hard-fibrous clover, alfalfa, etc. Constipation is, on the other hand, frequently produced by feeding materials such as bran, crushed corn, barley and muddy hay, which are rich in the salts of the earthy metals, and also upon the ingestion of sand (see clogging of stomach and intestines with sand), soil, wood and bark from trees when the animals are on an exceedingly scant diet (frequently observed during the World War). Behrens claims that obstipation is caused more frequently by damp, mouldy food, mouldy oats, fresh rye bread, food which is too fresh; and most frequently by very high temperature conditions, as is suggested by the fact that the disease is frequent during the warm season, particularly during May and June. (In cases of this kind it was probably a question of catarrhal intestinal spasm, at least the great frequency of obstipation of the small

bowel points to it). The prejudicial influence of these substances is particularly striking after a sudden change of food and if the animals do not have enough exercise, as for instance in army horses after the fall manoeuvers when their work has become diminished very greatly while they receive their customary quantities of food or even more. This explains the increased frequency of cases of colic among army horses after the fall manoeuvers to which Sustmann has called attention.

Retarded movement of the large intestines may cause obstipation of the gut, even on proper feeding. This is seen in old, enfeebled horses, in fat animals which do not exercise much, usually also in horses of a listless temper. Insufficient peristaltic motion may also be consecutive to chronic intestinal catarrh, and the latter may have developed upon a thrombotic or embolic basis.

The cause of the disease is sometimes a disturbance of mastication (bad teeth), because then the feed is not sufficiently broken up before it gets into the gastro-intestinal tract. Insufficient peristalsis and frequent anomalies of teeth in old horses explain the frequency of obstipation in advanced years.

The combined use of morphine and atropine against shoulder lameness causes many cases of obstipation with subsequent bloating or rupture of the stomach, because atropine diminishes the intestinal secretion and morphine suppresses peristalsis.

Secondary obstipation is seen after intestinal stenosis, after intestinal obturation existing for some time, in combined paralysis of the tail and sphincter, and exceptionally following thrombo-embolic processes of intestinal vessels if these have lasted at least for several days.

Pathogenesis. Retardation of peristalsis and a firmer consistency of the intestinal contents bring about, even under normal circumstances, a gradual accumulation of feces, particularly in the narrower portions of the intestines, where the removal of the contents is slow, even under physiologic conditions. An accumulation of feces occurs most frequently in the stomach-like dilatation of the colou, in front of the first portion of the small colou and it may spread from here to other portions of the colon and even to the cecum. Sometimes obstipation develops in the pelvic flexure and at other times again in the cecum in front of the comparatively narrow opening of the colon; also fairly frequently in front of the ileo-cecal valve, rarely, however, exclusively in the rectum, in the region of the second flexure of the duodenum and only exceptionally in the jejunum.

The accumulation takes place in either one of two modes. The above mentioned portions of the small intestines, most frequently the end piece of the ileum during or after one meal, rapidly become filled with coarse fibrous, dry feed (generally chaff). Since the gastric contents in horses enter the small intestines partly unchanged (Ellenberger), the feed mash, if insufficiently

broken up or if in a rather dry condition meets an impediment at the ileo-cecal valve, occasionally even in front of second curvature of the duodenum or in front of any curvature of the jejunum. The development of obstipation in the large intestine which is much wider, occurs, however, much more slowly. The retardation of peristalsis, the firmer consistency of the intestinal contents, the greater mass of the intestinal contents, cause an increasing delay of their transport and they become more and more desiccated. The appetite of the animals has not suffered in the mean time, and larger and larger masses accumulate in the affected portions of the large intestines.

Sudden or rapidly occurring closure of the small intestines, with subsequent stretching of the intestinal wall by the accumulated masses of feces, stimulate the occluded portion and those anterior to it to convulsive contractions which produce colicky pains. The contractility of the muscularis of the large intestine, however, diminishes from the start only very gradually since the accumulation and the closure are brought about very gradually, in the course of several days. Convulsive contractions and colicky pains, therefore, do not occur or only very moderately, and they are generally localized at a point in front of the obstipation. The dilatation of the filled portion of intestines, however, produces a disagreeable feeling of tension or fullness in the abdomen, which in combination with the absorption of intestinal poisons causes an intoxication which in its turn produces dullness of the sensorium. An exception is presented by those rare cases where obstipation in combination with a change of feed will produce lively gas formation in front of the closed portion, especially in the cecum. Then the irritating gases will produce strong convulsive contractions of the muscularis.

According to whether the transportation of accumulated feces is stopped suddenly or within a short time, or whether as in obstipation of the large intestines, the cessation occurs very gradually, contractions of intestines behind the obstructed portion cease after a few hours or only after several days, then the periodical filling of the rectum likewise ceases. An exception to this rule sometimes occurs in obstipation of the cecum in those cases where the desiccated fecal masses do not reach up to the cecal-colonic juncture and where the contents of the head of the cecum can be pressed partially into the colon.

The dried masses of feces may subsequently cause necrosis of the epithelial covering of the mucosa, and may in this manner lead to enteritis, or even to rupture of the wall of the gut. The decomposition of the thin-fluid feces accumulating in front of the point of obstruction in the course of time causes moderate bloating, possibly even enteritis; since there is no fermentation in the obstructed portions of the intestines, bloating will not occur there (absence of fermentation in the desiccated contents) excepting the peculiarly shaped cecum, at

base of which, in case the contents are dried to a lesser ree, not infrequently a plentiful accumulation of gas will e place. Obstipation of the small intestines and not infrently also obstipation of the large intestines is followed by econdary dilatation of the stomach (mechanical and reflex ibition of the emptying of the stomach). Enteritis which y have developed in the meantime and other complications se general symptoms (in cases of obstipation of the large estines only after several days).

Anatomical Changes. The obstructed parts of intestines dilated, their surface may be smooth or nodular, their ındant contents appear more or less desiccated, mortar-like; contents of the large intestines may even show the contours the pouches. The mucosa shows blood extravasation; it looks if covered with bran in consequence of epithelial necrosis, l sometimes shows larger patches of necrotic tissue. In more longed obstipation of the cecum one sees hypertrophy of the scularis and considerable chronic dilatation of the intestines. netimes a rupture is found at the place of the obstruction immediately in front of it; if there has been dilatation of the mach there may be rupture of the stomach.

Symptoms. In the most common type of **obstipation of the ge intestine** one sees in the beginning retarded defecation ıch may last for several days, then there is complete absence it in spite of repeated efforts. Obstipation of the cecum ers an exception because there may be defecation, though perps deficient, which only ceases in the further course of the ection. With the absence of defecation symptoms of colic pear, at first of a mild type and at long intervals, often lasting hours. Restlessness later on becomes somewhat more connous and intense, but does not reach a high degree. The mals lie fairly quietly on the floor, only occasionally turning ir heads toward the abdomen (rolling is rarely observed); y paw from time to time with their front feet and move their s. Squatting on the haunches may occur in any form of tipation. Not infrequently the patients place their feet like le horses in urination, so that the front legs are placed much ards the front and the hind legs much backwards, while the k is stretched out.

The abdominal circumference remains unchanged for some e, and occasionally until the disease has run its course, parılarly if the horse has a tense abdominal wall. The percusı sound is usually dull over the colon, and the dullness may end over the cecum; often, however, percussion may not re-l anything abnormal in spite of extensive accumulation of es, because the loops of intestines filled with firm fecal masses y not be in touch with the abdominal wall. The intestinal nds always occur less frequently and they may be suppressed irely in the further course of the disease.

Rectal examination reveals dilated, semi-solid, soft or entirely firm loops of intestines. If there is an accumulation of feces in the colon, which is quite frequent, one finds the whole left half of the abdominal cavity filled with the left portions of the colon, the lower one being characterized by longitudinal bands and pockets and being felt towards the median line, the smooth upper portion becoming larger towards the thorax, and the two other portions in the entrance of the pelvis leading to the

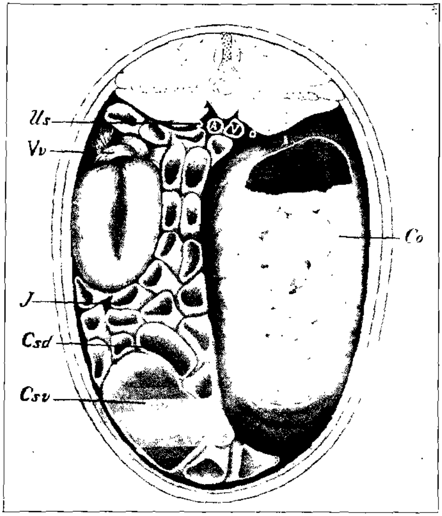


Fig. 14. Obstipation of the cecum and volvulus of the small intestines in a horse. (Oblique cross section of the abdominal cavity through the 4th lumbar vertebra and the flank.) *Us*, left ureter; *Vv*, place of volvulus of a greatly bloated loop of intestines; *J*, jejunum; *Csd*, left superior, *Csv*, left inferior division of the colon; *Co*, the greatly dilated cecum filled with feces, its base being distended by gas.

smooth pelvic flexure. The latter frequently becomes displaced entirely into the pelvis or slides towards the right side. If this is the case one can feel the longitudinal bands running from left to right, possibly with a spiral twist. Not infrequently the left upper portion of the colon is displaced to the right or to the left, beside or even below the dilated lower portion, but without the production of a true torsion. The enlarged stomach-like dilatation of the rectum may also be palpated in horses

unless they are large; it can be felt in front of the cecum, a little to the right of the median line, and it has the shape of a large semi-spherical, tough body, moving synchronously with the respiration, possibly covered by the anterior mesenteric root. The beginning of the small colon may be felt under the anterior pole of the left kidney, as a sausage-like body of the thickness of an arm, running obliquely from right to left. The cecum, filled with feces, is detected when the hand is directed to the right flank; it is recognized by its form and by the course of its two longitudinal bands; it may be as firm as the colon under similar conditions or its contents may even be almost as hard as a rock. The head of the cecum and the small intestines are frequently bloated in cecal obstipation. Loops of the small colon in which feces have accumulated are felt, by the hand introduced into the rectum as sausage-like loops, at the entrance of the pelvis and particularly to the left, externally or internally to the left portion of the large colon; they are provided with one longitudinal band and filled with balls of fecal matter.

The pulse and respiration remain normal for a long time, even for days, or if abnormal at all they present only slight changes from the normal. The appetite may be good in the beginning of the affection or for several days, even after the first symptoms of colic, the animals ingest some feed and this aggravates their condition and the restlessness after each meal. Later, however, the appetite becomes permanently abolished.

In a more advanced stage, usually only after several days, there is elevation of temperature, with debility and accelerated pulse, indicating the advent of complications (enteritis, peritonitis, meteorism, dilatation of the stomach).

E. Bauer has made interesting observations concerning indican in the urine in obstipation. In all cases examined an increase of indican was found: in obstruction of the cecum three to four times the normal amount, while in obstruction of the colon a rather moderate increase was usually found. If the accumulation of feces could be removed from the entire intestinal tract, the amount of indican became diminished below normal, but it remained high if the cecum could not be evacuated, even if diarrhea had been produced. Redecha failed to find a very marked regularity in the variations with respect to the quantity of indican.

The symptoms of **obstipation in the small intestines** vary from those in obstipation of the large intestines since they come on suddenly, within a few hours after food ingestion, or directly after the latter, if the duodenum is involved. The symptoms of colic are decidedly more marked, sometimes quite severe, and a stretched position of the animals, as in urinating of male horses, is observed. Defecation ceases within a few hours. Rectal examination reveals the extended ileum as a smooth cylindrical mass the size of an arm, situated in the plane of the posterior pole of the left kidney and to the right of the vertebral column running from above obliquely downward and backward

and to the right, or on the contrary from below on the left upwards and to the right towards the base of the cecum and continuous with the latter. The obstructed duodenum can likewise be felt as a cylindrical smooth body, the size of an arm, extending immediately behind the anterior root of the mesentery in a curved direction from the right to the left and adherent to the mesentery only by a short band.

In contradistinction to what happens in obstipation of the large gut, acceleration of the pulse and respiration appear one-half day or sooner after the occurrence of obstipation of the small intestine. This is obviously due to a secondary dilatation of the stomach or to early inflammatory changes of the mucosa, the pulse in particular rising to sixty and more per minute even on the first day of the disease. The appetite is completely suppressed.

Complications are not at all rare in either form, but are more common in obstipation of the small intestine. In the latter, that is, in one-third to one-half of the cases, secondary dilatation of the stomach (see page 138) occurs, although this condition is not at all rare in obstipation of the large intestine. Dilatation of the stomach may lead to rupture of the stomach. Rupture of the intestines, which is tolerably common in obstinate and unyielding intestinal obstipation and which always occurs in very extensive fecal accumulation in the cecum, like rupture of the stomach, leads to collapse (see page 142), or if the tear is not very large and the shock has not been too great, it will subsequently be followed by general acute peritonitis. A not infrequent complication in obstipation of the small intestines is enteritis.

Course. Obstipation of the large gut develops, as stated, very gradually; restlessness sets in only after a few days and the condition becomes worse by and by. Cases are seen occasionally where, after several days, the disease ends in recovery without having led to symptoms of colic. Fecal accumulation in the small intestine causes sudden attacks of colic and usually lasts one to two days. Obstipation of the large intestine always lasts several days, even two to three weeks (especially **obstipation** of the cecum).

A recurrence of the attacks of colic is not infrequently observed, either because errors of diet have not been corrected or because the intestinal muscularis has been weakened while the obstipation lasted, or because the accumulated feces have not been removed completely. The two latter circumstances are particularly effective on acconnt of anatomical conditions in obstipation of the cecum; and this form of fecal accumulation is seen not infrequently in the form of recurring attacks (socalled habitual, chronic or periodic colic).

Diagnosis. Retardation followed by cessation of defecation, the history of the case, and the usually mild attacks of

colic occurring at long intervals, the absence of general symptoms make a diagnosis of obstipation in the large intestine quite probable; however, only rectal examination can make the diagnosis absolute; its differentiation from similar affections, and its exact localization depend upon rectal exploration. And in this type the general symptoms usually come on sooner. Valuable assistance in the diagnosis and prognosis may be obtained by the quantitative determination of indican as proposed by Bauer. It is absolutely impossible to determine the seat of the obstipation from the behavior of the animals as suggested by Klemm.

The size and the firm consistency of the affected parts of the intestines, which can be ascertained by rectal examination, distinguish the disease from various other forms of intestinal occlusion, with the exception of stenosis or obturation of long duration, or of paralysis of the rectum; these can be differentiated by the different nature of their onset and by the fact that the site of the stenosis or of the obturating foreign body can be felt from the rectum. In affections of the stomach and in obstruction of the small intestine, the contents of the large intestines likewise become desiccated if the disease has lasted for any length of time; however, the large intestines are not dilated by the desiccated feces but are diminished in diameter.

Prognosis. If proper treatment is instituted in good time, the great majority of cases end in recovery. The longer the disease has lasted the firmer the stagnating intestinal contents are, and the more dilated the obstipated intestines the less hope there is to remove the obstruction. One also must always consider the exact site of the obstipation, because if it is in the small intestines or cecum, a fatal issue is comparatively frequent, and recurrences often occur. In obstipation of the cecum the reappearance of defecation does not always indicate improvement, because masses of desiccated feces may remain behind in the cecum. If complications have already occurred there is no hope for recovery, except in secondary dilatation of the stomach, which can be treated successfully.

In the Budapest Clinic the mortality for different years ranges between 3 to 10%. Behrens gives a mortality of 3.8% for obstipation of the small bowel, and of 4.7% for obstipation of the large bowel. The figures given by Frese are 4.6% for the former and 27.7 for the latter. Brieg observed at the Copenhagen Clinic a mortality of 6.44% in obstipation of the colon, 17.97% in obstipation of the cecum and 67.65% in obstipation of the small intestines. At the Vienna Clinic Wirth in 10 years had a mortality of 14.3% (in 6 years with an average quality of feed, 63%; with feed of a poorer quality, 34.3%; and with very poor conditions of feeding, 44.3%). Wilson observed a mortality of 60% in obstipation of the cecum.

Treatment. To remove accumulated masses of feces from the large gut, those which can be reached manually from the rectum should be evacuated, then a rectal injection of a large

amount (30-40 qts.) of lukewarm water should be given, being allowed to run in under a low pressure. The injected water is partly soon expelled, but Dammann and Marek have shown experimentally that in horses without obstipation the fluid may travel up to the middle or even to the beginning of the colon, and that it may therefore be expected to produce softening of the feces in obstipation. Cold water is not well adapted for this purpose because it stimulates the intestines to contract and to prevent the progress of the fluid inward. Accumulations of feces in the cecum or in the small intestines cannot be influenced even by very large quantities of water.

In order to soften the fecal masses which are situated nearer to the stomach, and to stimulate peristaltic movements, neutral salts are administered with much water and in large doses (700 to 1,000 gm.) with much water, or, better, in a 5% solution: that is, 8 to 15 liters of fluid, with the stomach tube, a procedure with which Wirth has recently obtained very favorable results (solution of the obstruction after the fourth to sixth infusion of 10 to 15 liters of a 5% solution of sodium sulphate, or, if necessary, of the same quantity of pure water 3 times daily). In cases of obstipation of the cecum, colon and rectum, aloes (30 to 40 gm.; according to Hoehne, 50 gm., at intervals of 3 days for a period of 2 weeks) are very effective, also extract of aloes (15 to 25 gm.). After this treatment the animal should be given a day's rest, even after the colic pains have ceased, in order to prevent profuse and sometimes unmanageable diarrhea. Toepfer, Mayr, and others found istizin (a preparation of synthetic anthrachinon) to be an excellent tasteless and odorless laxative and a substitute for extract of aloes (for small horses 6 to 10 to 12 gm., larger horses 15 to 20 gm., very heavy horses 20 to 25 to 40 gm., in capsules; action at the end of 30 to 50 hours and continuing from 1 to 2 days; together with arecolin after 12 to 24 hours). Castor oil is well adapted for all cases (300-500-700 gm., best in the crude emulsion or with ½-1 quart of a neutral oil; may also be given with 50-70 gm. of ether, see page —). If the cases are not unduly severe, copious defecation occurs promptly and is followed by recovery. In severe cases the discharge of the fecal masses, which have been softened by the rectal injections and the neutral salts, requires further remedies by which the bowel is stimulated to vigorous contractions. Not earlier than half a day after the injections have been given, and the neutral salts or castor oil have been administered (few hours if the impaction is in the ileum), subcutaneous injections of eserine (0.06-0.08 gm.) with pilocarpine (0.12-0.25 gm.) or with arecolin (0.05-0.08 gm.) are of service. As a complete substitute for arecolin, cesol (methylate of nicotinic acid-methylester) may be used, according to the observations of German veter-

inarians (Holzmann, Findeisen, Krueger, Burchard, Rosenthal and others). It may be obtained in a 50% slightly yellowish, sterile solution in sealed ampules containing 7.5 cc. It is administered in single doses of 7.5 cc., or, according to Burchard, better in 12 cc. (15 cc. at the most), intramuscularly. (Long-continued, painful swellings follow subcutaneous injection.) In case of necessity the injection is repeated in 15 to 30 minutes. In such cases Rosenthal injects, instead of a second dose of cesol, 0.5 gm. barium chloride, intravenously. In place of cesol, neu-cesol (yellowish, brown solution) in 1-gram doses may be administered with the same effect. Exhaustion, strangulation and eructation, which are sometimes observed following the administration of arecolin and pilocarpin, were not observed when cesol or neu-cesol were administered. Without previous softening of the fecal masses, drastic drugs, such as barium chloride or eserin in full dose, could easily rupture the intestines. Since the drastic remedies aggravate the abdominal pain, the animals must be watched and prevented from throwing and rolling, which might also give rise to rupture of the gut. In most cases the remedies must be repeated several times, and Paetz suggests for this purpose small single doses (0.025 gm.) of arecoline which are administered at intervals of ½-1 hour, or at intervals of one to several hours. This last mentioned gentle method of treatment with drastic remedies is deserving of consideration not only in mild cases but also in severe obstipation.

The effect of different forms of treatment is assisted by massage or kneading of the masses of accumulated feces from the rectum (Sobelsohn), this further causes the intestines to contract and the fecal masses which can be reached may be broken up or moulded. Massage alone is usually not successful, except in obstipation of the small intestines if the jejunum can easily be reached by the hand introduced into the rectum. Hummerich and Kalcher obtained good results in obstipation of the large intestine by rolling the animals on their backs (see page 218).

The treatment of impaction of the cecum by an induction current, as recommended by Bromberger, seems without significance, according to Frese, in spite of the fact that Bezel has recently reported favorable results in light cases of impaction or in colic free from complications. Bromberger's method consists in fastening two large electrodes to the lumbar spinal column and in the region of the apex of the cecum, and gradually increasing the current until the abdominal wall begins to tremble or becomes hard as a board, for a period of 15 to 20 minutes.

In very severe obstipation of the ileum which does not yield to any of the forms of treatment described, one might try laparotomy to remove the desiccated contents through the left sided laparotomy wound, either by propelling them by hand or by taking them out after enterotomy. In obstipation of the large intestine, laparotomy does not appear indicated, because one cannot remove the great masses of desiccated feces completely and the sutures would not keep after partial removal, being torn by the masses which have remained behind. The cases of Gaullet and Hobday prove conclusively the danger of laparotomy in obstipation of the large intestine. Deghilage has massaged intestinal loops through an incision made into the upper vaginal wall of a mare and has in this manner brought about recovery.

Secondary dilatation of the stomach can only be treated by the use of the stomach tube (see page 145). This instrument should always be used in obstipation of the small intestines, even before symptoms indicate dilatation of the stomach. If the gastric tube is used repeatedly under these eonditions, rupture of the stomach can usually be prevented.

Obstipation in the large intestine also calls for dietetic treatment. Instead of rough feed or grain, which horses with obstipation of the large intestine take by preference for some time or after a temporary improvement, but which will surely lead to an aggravation of the affection, the animals should receive juicy food stuffs, like bran or flour gruels, beets, bulbous plants, green soft feed, always in small amounts and at intervals. The animals should be prevented from eating bedding straw by the use of a muzzle or otherwise. Pasturing the animals is very good. However, they should not be fed on any bloating green feed, particularly not in obstipation of the cecum.

Prophylaxis. Animals inclined to suffer from obstipation should be fed with fresh hay and green feed or molasses, or they should be pastured at certain times; if this cannot be carried out, the systematic administration of salts with the feed may properly be substituted to a certain degree. Faulty teeth must be repaired. The predisposed animals should be subjected to close observation, so that one can take the proper steps whenever there is any disturbanee in defecation. Ducasse recommends in such cases the injection of pilocarpine followed by eserine, but it would be preferable to administer small doses of arecoline. For the treatment of army horses, Sustmann advocates a slow return, on foot, from the manoeuvers instead of by the short railway trip, because in this manner the animals are gradually accustomed to the diminished amount of work. The attempt should be made to secure permanent hay rations for army horses.

In order to prevent poorly nourished animals from eating foreign substances, an increase in the amount of roughage and protection from cold to decrease the quantity of necessary nutriments are very serviceable. Horses that are suspected of having eaten such substances should be given a laxative even before disturbance of health become manifest.

Clogging of the Stomach and Intestines of the Horse With Sand, Etc. (Sand Impaction, Sand Disease, Kumgata, Saburra). This condition is the result of a continuous consumption of sand, mud, slag, small stones, particles of soil, coal, etc. In a practical sense disturbances of health due to the consumption of wood and bark also belong in this category. The involuntary consumption of such material with the feed or the drinking water is frequently due to feeding sandy, muddy or dusty feed, to pasturing in sandy regions with scant turf or with sand accumulated in the grass after stormy weather, feeding from the ground, picking up grain from the ground, consumption of litter and manure in stalls with wooden or dirt floors. On the other hand large amounts

of foreign material are taken up with the water by drinking from shallow brooks or pools, swollen brooks and rivers, cisterns with sandy or muddy sediments, and draw wells with low water level. Independently of feed and drink, horses are often led to consume such material voluntarily, usually in the stall, or under special circumstances (as in the World War) in the open, by continued sensation of hunger, by too limited feeding in general, but also by scant ratious of roughage and of bulky material. Primary pica may play a role only rarely, as probably in remounts (sand-eating in remounts).

Sand disease has been known in the Kirghiz steppes for a long time (Wedernikow), but attained an especially wide distribution and significance during the World War, particularly in the field army in Volhynia and Macedonia (according to Orgeval nine-tenths of the horses of the southern army of the Entente powers suffered from it).

Anatomical Changes. The sand is found in both the stomach and intestines (Wedernikow), or mostly only in the intestines (Gnuechtel, Orgeval, Robin, Kalusch and others), and here particularly in the stomachlike dilatation, the pelvic flexure, the transverse layer and the lower layer of the colon. In the stomach stony sand concrements up to the size of a head and weighing 2 to 5 kgs. may be found. The impacted portion of the intestines is enlarged and filled with masses of inspissated fecal matter mixed with sand or even concrements, and the contents may weigh from 30 to 40 kg. Not infrequently the intestinal mucosa is found to be covered with a more or less extensive layer of sand or mud, which inhibits absorption and secretion of the mucosa. In lingering cases the mucosa of the stomach and intestines shows no changes whatever, whereas otherwise it shows streaky, patchy or more diffuse reddening, that is, catarrhal or inflammatory changes. Perforation or rupture of the intestines may also be encountered.

Symptoms. The first symptoms appear, according to Gnuechtel, after 2 weeks; according to Haversang, under certain circumstances after months; according to Kalusch, after an average of 4 to 6 weeks; in well-nourished animals with partial passage of the consumed sand in 8 to 12 weeks, while under unfavorable feed conditions colicky symptoms are said to appear in heavy horses after the accumulation of 35 kgs. of sand, in large horses after the accumulation of 30 kgs., and in small horses after the accumulation of 20 kgs. of sand. On the other hand, Bethcke observed in one barn the first case of colic 5 days after slag had been spread in the stalls as the lowest layer of the litter, and after the consumption of a total of 5 lbs. of slag (mostly small gravel, besides particles of coal, small rocks, small pieces of glass and porcelain, pieces of wire, pins and wire nails). With a good appetite there will first be noticed a slight reddish yellow discoloration of the conjunctiva, frequent yawning and symptoms of pica; then a gradual increase in the appetite until it becomes voracious. Attacks of colic follow in about the third week; at first they are of a light character, especially with changes of feed and weather, but later they become more frequent and severe. Simultaneously the animals appear very weak and become very much emaciated. Death follows in 1 to 2 months. In other cases there is observed at the beginning weakness, permanent recumbency, light colic, inappetence, diarrhea or obstinate constipation with a medium degree of bloating, and sometimes polyuria; furthermore, a medium acceleration of the pulse and respiration as well as light fever (38.5 to 39.5° C.,

101.3 to 105° F.). According to Gnuechtel cases are sometimes observed that take a slow course without colicky symptoms, but show progressive emaciation and loss of strength in spite of a good or even voracious appetite and good feeding to the end; toward the end there is accelerated pulse and feces are formed into small balls and show a glistening covering; death comes from exhaustion.

The diagnosis is based on the detection of sand or other foreign material in the feces (often repeated systematic examinations are necessary), the frequent appearance of colic, the result of rectal examinations (peculiar consistency of the diseased portions of the intestines), and especially upon the history of the case. Sometimes the presence of sand will even be recognized upon puncture of the cecum (Orgeval).

Treatment. In general the treatment is similar to that usually followed in impaction. The respective drugs and other treatments, however, should be repeated several times. Stronger laxatives should not be given subcutaneously until the evacuation of foreign material in the feces has already begun. (Kalusch had bad results from the use of arecolin.) During convalescence succulent feed or feed containing much water should be given for 3 to 4 weeks, since some of the sand will remain in the intestines for weeks.

Prophylaxis. To remove the cause it may be necessary or advisable, according to conditions, to provide pastures or green feed; to increase the rations, when necessary by the addition of a filler (hay, straw); to clean the feed of sand and dust by shaking, etc.; to keep clean the feeding places in the open; to cover the front end of the stall with wire netting; to dig a correspondingly wide and deep pit in the stalls and fill it with manure or compost; to muzzle the animals between feeds and when at work when they are left standing in the open for a long time; to provide clean drinking water, if necessary allowing it to settle; to prevent the drinking of open water, and to supplement the feed with lime preparations if necessary. Under certain conditions it may be advisable to treat all the horses in the barn with laxatives.

(b) Obstipation in the Intestines of Carnivora.

Occurrence. Accumulation of feces in the intestines is common in dogs, much rarer in cats. Fröhner, in 70,000 sick dogs during the years 1886-1894, saw obstipation in 2% of the patients. In the Budapest Clinic 2 to 3% of the dogs had obstipation; in 1903, exceptionally, 7%.

Etiology. Obstipation is frequently produced by hard bodies, especially fragments of bone, more rarely fruit stones, pebbles or portions of soil which interfere with the passage of the feces. Dry feeding, exclusive or abundant vegetable feed (bread, dog biscuits, leguminosæ, flour paste) or exclusive feeding with bones, produce dry, mortar-like feces which can be moved along only slowly. Hairs, blades of grass, etc., which form firm masses in the feces act similarly. Obstipation, due to swallowed hair, occurs not infrequently in cats.

Retarded peristalsis is the cause of obstipation when it occurs in consequence of lack of exercise (chained dogs, house

dogs), or when it is seen in older, debilitated animals. Chronic intestinal catarrh may also lead to insufficient peristalsis.

The disease occurs as a secondary affection in consequence of stenosis or obturation of the intestine, of painful conditions of the abdominal muscles (rheumatism, pachymeningitis), or of the neighborhood of the anus (inflammation of the anal glands), or after matting of the hairs around the anus. Obstipation is also usually present in chronic diseases of the cord.

Anatomical Changes. Feces collecting in some portion of the intestines, usually in the colon or rectum, form very dry, mortar-like, dark brown lumps. There are cases in which such lumps of feces form firm cylinders as thick as an arm, which fill the whole of the large intestine (Kitt); exceptionally the whole intestinal tract may be filled with similar masses of feces (Fröhner). If the affection lasts longer, necrosis, hemorrhagic or diphtheritic inflammation and even perforation of the intestinal wall may develop.

Symptoms. In spite of repeated efforts the animals cannot defecate or they void small dry lumps of feces, the surface of which is occasionally covered abundantly with mucus or blood; this occurs when the mucosa has become inflamed or has been injured. Sometimes, in spite of existing obstipation, the animals void a thin-fluid, very fetid stool; this occurs when the obstipation mass has become softened at the periphery or in the center, so that the fluid contents from portions of the intestines nearer to the stomach can pass by.

The abdomen is sometimes drawn in, sometimes bloated, and the abdominal wall is then tense. On palpation of the abdomen one feels in front of the pelvic inlet, below the vertebral column, and parallel to it a cylindrical, firm, sometimes hard mass, variable in length (socalled fecal tumor, fecal cord). The mass may be felt behind the liver and sometimes even further up on the right side of the abdominal cavity and it is freely movable. In some cases, however, only one or several, occasionally very large lumps of feces are found.

Sometimes swelling and reddening of the neighborhood of the anus can be observed. The finger introduced into the rectum feels hard fecal masses; sometimes also fragments of bone; if a rectal speculum is used one sees the lumps of feces, also a dark red discoloration of the mucosa, which may be covered by gray membranous deposits.

The repeated fruitless efforts cause anxiety and pain expressed by whining. In other cases the animals only show listlessness and sluggishness. Their gait is stiff, they hold the tail straight or strongly curved at its root.

The appetite may be preserved during the first day and this aggravates the condition. The appetite diminishes, how-

ever, in the further course, and finally disappears while the thirst becomes increased. Vomiting occurs exceptionally and particles of fecal matter may be expelled.

The temperature remains normal for days, but if enteritis has followed upon obstipation the temperature becomes elevated and septicemic fever may finally set in.

Course. Obstipation of a not too severe type may be overcome by the animals' own repeated efforts and, provided that they have not been too long neglected, even grave cases, will end in recovery if the proper treatment is instituted. If the disease is left to itself after it has arrived at a later stage, there will be progressive deterioration until enteritis, septicemia, peritonitis or occasionally uremia (in consequence of compression of the first portion of the urethra), close the clinical picture. If the morbid affection has lasted a longer time, i. e., two to three weeks or longer, even the proper treatment cannot save the animal any more, on account of the gangrene of the intestinal mucosa which has occurred.

Diagnosis. Constipation in spite of fruitless efforts, combined with the detection of desiccated fecal masses in the rectum or colon, point to the correct diagnosis. If the animal has previously been well and if the accumulation of feces can be explained from the character of removed fecal masses, one may assume primary obstipation. Otherwise a careful examination of the organs is necessary in order to determine whether we are not dealing with a case of secondary obstipation. Chronic diseases of the spinal cord have to be considered particularly, because these often first attract our attention through obstinate obstipation. Diseases of the region of the anus and of the pelvic organs can be recognized easily on the basis of the history and as the result of palpation (sometimes to be carried out during narcosis). After long continued starvation or preceding diarrhea, defecation may be infrequent, but in such cases there is no accumulation of feces.

Treatment. If obstipation has not lasted long and if the lumps of feces which can be felt from the rectum are not very hard, the local treatment may be confined to the introduction of large masses of water into the rectum; this softens the lumps of feces, makes them slippery and stimulates the rectum to contract. One may use pure lukewarm water, better soap suds or water with oil, or pure oil. Thin fluids are allowed to run into the rectum from an irrigator or a funnel provided with a rubber tube; low pressure must be used. The softening of the feces may be hastened by cautious pressing and kneading. This simple procedure, which must eventually be repeated, usually suffices to remove the morbid condition. In mild cases glycerin enemata are sometimes sufficient.

When the obstipation has lasted for a considerable time, and when mortar-like masses or fragments of bone are present in the rectum, they should be removed with the finger or with a pair of forceps; then the more anteriorly situated, usually less hard masses, can be softened and removed with lukewarm water irrigations; in such advanced cases massage is not indicated. The artificial removal of fecal masses and rectal irrigations sometimes have to be repeated for several days, until it has finally been possible to remove piece-meal a fecal cylinder which may have been 20-30 cm. long. In these cases massage is no longer appropriate, either because the intestinal mucosa may be affected or because a traumatic injury to the intestinal walls might be produced.

Laxatives should be used only in recent cases or after the mechanical removal of the fecal masses near the anus, because if used in old cases by themselves alone, they may make the condition worse. The prescriptions to be employed are: Castor oil (1-3 tablespoonsful in an emulsion with 1-3 to 1-4 parts gummi arabicum and 5 parts of water, or in gelatine capsules 3-5 gm. each), calomel (dogs 0.20-0.30 gm., cats 0.1-0.15 gm.), aqua laxativa viennensis (50.0-100.0 gm.), occasionally with the addition of sodium and potassium tartrate (5-10 gm.), extractum cascaræ sagradæ (2-10 gm.), phenolphthalein (10 gm. for large, 5 gm. for small dogs). The neutral salts (see page 360) may likewise be used, and in very obstinate cases drastics may be employed (1-5 drops of croton oil in 10-30 gm. of castor oil); also tubera jalapæ (0.2-0.4 gm.). Drastics must be reserved for those cases where no inflammation of the intestinal wall is present and where fragments of bone or other hard bodies have not penetrated into the wall of the gut.

Where accumulation of feces is due to a tumor, an abscess, an enlarged prostate, a cicatrix, surgical interference is necessary.

The diet must be regulated so that the patients do not receive any food containing flour (bread, dog biscuits, potatoes, vegetables) or bones, but exclusively fluid food, such as broth, soup, milk. Animals which are predisposed to obstipation should receive as little of carbohydrates as possible, and they should be exercised frequently.

(c) Impaction of the Intestines of Ruminants.

Occurrence. Double mastication, preliminary preparation and mixing with abundant fluid in the fore-stomachs of ruminants make primary fecal impaction of rare occurrence in these animals. The disease has been seen in cattle and in goats.

Etiology. The occurrence of primary impaction in ruminants generally depends upon *continuous feeding with undigestible, dry feed* (leaves or matted hay). Moist mouldy straw,

withered dry and rotten cabbage may also be the cause of the disease. Mathis saw grave cases of fecal impaction in cattle after long railroad transportation.

Symptoms. The disease is manifested by obstinate constipation, by moderate bloating after each meal, however without the accumulation of feed masses in the rumen, gradual decrease of appetite and retardation of rumination. Bruce observed several cases of sickness and death in sheep 2 weeks after they had been fed alfalfa hay which had an admixture of some cactus plants and sand. Signs of restlessness are either lacking or are very insignificant. The feces are dry like peat, sometimes covered with a good deal of mucus and are voided in small amounts in spite of efforts. The intestinal sounds are absent or weak. Rectal exploration reveals, in the region of the right flank, sausage-like loops of intestines filled with firm feces; in small ruminants the desiccated feces may be felt through the abdominal wall. Besides constipation, Bruce observed in sheep depression, restless movements as though the patient were blind, head bent to the side or downward, gritting of the teeth, in severe cases constant recumbency, and finally death in coma after 10 to 21 days, after the feces had become soft and evacuated with admixture of blood and sand. As a rule only very severe cases terminate fatally (Joehnk observed rupture of the intestines in a cow), while usually recovery takes place within a week.

Diagnosis. Displacements of the intestines can be distinguished from impaction by the severe symptoms of colic and by a rapid deterioration. Stenosis of the intestines can often be differentiated only by rectal exploration.

Treatment. Injections of large quantities of water and the administration of neutral salts (500-1000 gm. for cattle, 50-100 gm. for goats) are indicated; also aloes (40-60 gm. for cattle, 10-20.0 gm. for goats); istizin (cattle 30 to 50 gm., yearlings 15 gm., calves 2 to 5 gm., goats and sheep 3 to 5 gm., action in 15 hours), which, however, like aloes, may cause diarrhea in calves when their mothers are treated with large doses (Schwab). Also tartar emetic (10-20.0 gm. for cattle, 0.2-2.0 for goats) alone or in combination with salts (tartar emet. 15.0 gm., sod. sulph. and magn. sulph. āā. 500 gm., given in three doses during one day for cattle). Eserine, eseridine, pilocarpine or arecoline may likewise be indicated (see page 85).

The diet should be made up of potatoes, beets, bran or flour soups, green feed, leaves of beets.

Wildfeuer observed in an elephant impaction of the large intestine with tympanitis, following extended railroad transportation during cold, wet weather and the feeding of bran of a poor quality. The observed symptoms were periodic groaning, alternate raising and stretching of both right legs, fruitless

attempts at defecation, pronounced distention of the abdomen (particularly on the right side), metallic sounds in the intestines, and the presence in the rectum of two fecal balls the size of a man's head. After massage, the introduction of about 10 buckets of water into the rectum, the administration of 5 kgs. sodium sulphate and the subcutaneous injection of 0.2 gm. arecolin hydrobromide had no effect, and 50 gm. barium chloride placed between large pieces of bread was allowed to drop out of the mouth. The evacuation of feces and gases was finally accomplished after 2 hours following the injection of 50 gm. barium chloride (50 gm. in 3 liters of water with 250 gm. sugar) into the pharynx and simultaneous subcutaneous injection of 0.3 gm. arecolin and the introduction of water into the rectum.

(d) Impaction of the Intestines of the Hog.

Etiology. Hogs develop impaction of the bowel in consequence of exclusive dry feeding with grains or after ingesting short cut chaff, or after the ingestion of much sand if the latter has not caused enteritis. Sometimes continued stabling causes the affection.

Secondary impaction is frequently seen after chronic hog cholera or chronic tuberculosis, or after stenosis.

Symptoms. One observes diminished appetite, increased thirst, frequent grunting and efforts at defecation. In the beginning the animals are still able to press out some lumps of fecal matter, later on the constipation becomes complete. On palpation of the abdomen of not too fat hogs, one can feel large intestines filled with desiccated feces; occasionally one can detect adhesions of loops of intestines. Oppenheim saw in a hog with a high degree of impaction, retention of the urine, due to compression of the neck of the bladder.

Grave cases end fatally in consequence of enteritis.

Treatment. Abundant injections of water (2-4 qts.), manual or instrumental removal of the fecal masses accumulated in the rectum, followed by the nse of laxatives, usually relieve the obstipation. Neutral salts (20-50.0 gm.) are best added to tbe drinking water or powdered upon the tongue of the animal or conveniently placed upon the back of the tongue in pill form with dressing forceps or a similar instrument (see distomatosis of the liver), without fear that they will find their way into the cul de sac of the pharynx, as reported by American observers. In order to prevent the medicine from getting into the trachea when the pig squeals uninterruptedly during medication, American veterinarians simply place the liquid drugs upon the tongue with a spoon and allow it to run over the back of the tongue into the pharynx. Cathartics should be introduced into the stomach by the aid of the stomach tube (tartar emetic 0.5-1.0 gm., castor oil 50-100 gm., dissolved in water or mixed with it), or eserine (0.005-0.02 gm.) given subcutaneously. Tartar emetic, castor oil or calomel may be given in the shape of an electuary. According to Mayr and Schwab, istizin (1 to 2 doses of 3 gms.) gives good results (action after

26 to 48 hours). Dietetic treatment consists in feeding bulbs, fallen fruits, green feed, cut pumpkin, sonr milk or whey.

(e) Impaction of the Intestines of Rabbits.

Etiology. Rabbits develop impaction of the large intestine after too abundant or exclusive dry feeding, particularly after dry bran or undigestible food stuffs have been given continuously.

Secondary impaction occurs after chronic intestinal catarrh in the presence of enteroliths and sometimes in consequence of painful affections of the rectum or of the parts in the region of the anus.

Symptoms. There is at first retarded defecation, then complete constipation, diminution of appetite and listlessness. Palpation of the abdomen, which is very easy, reveals the presence of desiccated masses of feces in the large intestines.

If proper treatment is instituted early the disease ends in recovery.

Treatment. Repeated rectal injections of water, and soap suppositories are to be employed (from tea to tablespoonful doses). Syrup with milk in equal parts, pills of the leaves of senna (1-2.0 gm.) or of rheum (0.2-0.4 gm.), tincture of rheum (5-10 drops in water), also root of jalap (0.2-0.3 gm.), or calomel (0.05-0.2 gm.) may likewise be used.

The diet should consist in green feed, juicy roots, lettuce, etc.

(f) Impaction of the Intestines of Fowls.

Etiology. The disease is rare in domestic fowls which are kept in the open air; if it occurs at all it is generally due to foreign bodies (pebbles, feathers, animal parasites, pieces of rags). More frequently, although still rarely, the disease is seen in over-fed, highly bred fowls kept in confinement in cages, or in house birds. These animals suffer from a combination of heavy undigestible feed and sluggish peristalsis. Impaction in the cecum, due to improper feeding, appears in enzootic form, particularly among young turkeys. The matting together of the feathers around the cloacal opening may likewise prevent defecation.

Symptoms. The birds drop dry feces in small amounts at long intervals and with effort; this attracts attention since under normal conditions their feces are dropped with great ease and are soft and mushy in consequence of the admixture with urine. If obstipation has lasted for some time there is listlessness, depression, lack of appetite, and attention is attracted to the birds even if the irregularity in defecation has escaped notice.

If the disease is neglected it may lead to death in consequence of necrosis of the intestinal wall, and exhaustion. Bailer observed unexpected death in a chicken with accumulation of feces in one of the ceca.

Treatment. If there is a mechanical impediment it must at once be removed; matted feathers may be untangled after wetting them with water; desiccated masses accumulated in the cloaca can be removed with a spoon. To remove the feces contained in the rectum, the careful injection of olive oil or soap water is to be recommended; in small birds a dull sound dipped in glycerin or castor oil may be introduced into the rectum.

If the impaction is in some part of the intestines nearer to the stomach, castor oil (table or teaspoonful doses), rheum (0.4-0.6 gm. with butter or honey in pill form), or eventually calomel (0.05-0.2 gm.) may be used with advantage. Neutral salts may be given in weak solutions (1:200) with the drinking water.

The dietetic treatment requires green feed, soaked grains, boiled bran, lettuce, carrots, fruit.

21. Internal Closure of the Intestine. Obturatio intestini.

By obturation of the intestines is meant a sudden narrowing or a complete closure of the lumen of the intestine by some body situated in the bowel. The affection occurs almost exclusively among horses and carnivora.

Occurrence. Obturation of the intestines is a rare affection of horses. Among the horses of the Prussian Army this form of obstruction of the intestines was seen in seventeen years only in 0.3% of the cases of colic; in the Budapest Clinic it formed 0.2 to 0.7% of the cases of colic. (Holterbach claims that foreign bodies in the duodenum are not rare in cattle.)

Etiology. Obturation of the intestines in horses is usually brought about by enteroliths or lumps of fecal matter, more rarely by parasites, exceptionally only by foreign bodies.

Intestinal calculi (enteroliths) are mainly composed of phosphate of ammonia and magnesia (90%); they also contain some (½-1½%) carbonate and phosphate of lime, common salt, and chloride of calcium and have an average specific gravity of 1700 (Fuerstenberg). The most frequent cause of calculi formation in the intestines is the continued abundant feeding of wheat and rye bran (the disease is frequently found among the horses of millers and bakers), which contain an abundance of phosphate of magnesia. The latter dissolves in the acid contents of the stomach and of the small intestines, and is precipitated in the alkaline contents of the large bowel; it com-

bines with the ammonia formed during putrefactive processes, and gives rise to phosphate of ammonia and magnesia, crystallizing around bodies which may have accidentally gotten into the large intestine (grains of oat, pebbles, splinters of iron, swallowed tooth [Fobelot]). Zschokke, however, claims that intestinal disturbances play a considerable rôle in the production of intestinal calculi. The latter are formed in the large intestine, as a rule preferably in the stomach-like dilatation of the colon. We here usually fine one calculus, variable in size, generally more or less spherical, sometimes weighing over twenty pounds; there may exceptionally be several calculi, even many of them; in the latter case they are irregularly formed. Colin examined horses for calculi; he found them 23 times (2.5%) in 900 cases examined, always in the stomach-like dilatation of the colon, only once in the cecum. Calculi have exceptionally been found in the small intestine; their mode of origin could then not be explained; in some of these cases the calculi were probably gallstones, as pointed out by Lewin.

According to Fürstenberg a new layer is formed around intestinal calculi after each meal of bran. A calculus of 14 pounds with a diameter of six inches on section showed 720 concentric layers; it would, therefore, have required 360 days for its formation. In a case of Pastore a calculus of the size of a fist had been formed in less than a year.

Besides the true calculi, pseudocalculi (mixed forms, pseudoenteroliths) are also observed. These are formed by the ingestion of sand, dirt, wood, etc. (see clogging of stomach and intestines with sand, etc.). According to Robin, in case of the ingestion of wood there are formed calculi consisting of wood fiber pasted together as though consisting of softened cardboard. Or, as described by Loerret and Perreau, the masses may have the appearance of cold tar, or the foreign material may become pasted together with the contents of the large intestines into dry, mortarlike fragments and these form into larger masses. Phosphates and carbonates of calcium (particularly when bran is fed), ingested hair, dry vegetable parts or other foreign bodies may also be mixed in them (in the case of Deysine an ingested sponge had become encrusted with salts of lime). Likewise concrements of plant hair are often found (plant hair bezoar, phytobezoar), consisting of plant hair (hairs of oats) and vegetable fibers with the deposition of inorganic salts similar to the hair balls in the forestomachs of ruminants (see page 109; also note Fig. 15).

The surface of pseudo-calculi and concrements is usually uneven; they often have sharp corners or may be covered by a crust of phosphate of ammonia and magnesia. They are much lighter than genuine enteroliths.

In a case of Grimme several hair balls were found in the large colon of a horse which had been fed for nine weeks with oat meal; they were up to the size of a fist and had been formed from the hairs of grains of oats. Wiesner found in a three months' old foal a concrement, obstructing the terminal portion of the ileum, composed of food particles, hairs and infiltrating lime salts.

Bellac reports the frequent occurrence in some regions of France, especially in Brittany, mostly in yearling colts (in some

cases in 50% of the colts), of conglobates (fecal, feed and sediment balls), consisting of matted clover and **Juncus** (rush) fibers, following the feeding of green clover before it was in bloom (mostly in April to the beginning of June). At this stage the thin, elastic fibers of the clover plant are not thoroughly broken up during mastication, and the same is true of the fibers of the rushes at all seasons of the year but especially in winter. In animals with abnormal teeth the insufficiently broken up stalks of hay, straw, and parts of other roughage are sometimes found closely matted together into spherical or short cylindrical balls from the size of a fist to that of a child's head. Such fecal balls occur in the colon, where they are frequently found singly

Fig. 15. Pseudointestinal concrements from the small colon of a horse (removed by laparoenterotomy).

and occasionally several are found. In new-born colts the meconium may sometimes occlude the intestines (Toepper).

The lumen of the small intestines may be obturated by animal parasites such as ascaris, gastrus, larvæ (Kater, Rexilius).

The anterior portion of the small intestine is only rarely obturated by swallowed foreign bodies (Bech, Jacobin and Clare, Angebauer, authors' observation). Closure by a hematoma formed in the intestinal wall is also quite rare (Uhlig, Kitt, Schleg & Johne).

Obturation of the intestines of **dogs** is often produced by foreign bodies swallowed in play or in retrieving (see page 152); also by pieces of bone and cartilage, occasionally also by hair balls, lumps of feces, parasites (tænia). In the case of Jakob a 6-months-old dachshund had swallowed a knotted rope about 6 meters long. Similar foreign bodies are found much less frequently in cats.

In **ruminants** and **hogs** obstruction of the intestinal lumen is sometimes (though infrequently) brought about by hair and fecal balls or by conglobates or plugs consisting of stalks of grass (Thum), ingested foreign bodies (in Otto's case in a pig by a long branch of a shrub), or by a hematome on the intestinal wall (Eber). (Fetting found in a young heifer, which had died of digestive disturbances, a young cat wedged in the small intestine; Wyssmann saw a case with obturation of the large intestine by masses of fibrin; Spörer saw a case where a potato had become wedged in the small intestine and one case where a vegetable stem formed the impediment.)

Pathogenesis. Foreign bodies formed in the intestines (calculi), or arrived there from without, may sometimes remain in a wider portion of the intestinal tract without doing any harm at all. Zschokke found, in a miller's horse which had always been well, forty-two pounds of intestinal calculi, one alone weighing twenty pounds. At other times calculi are the cause of chronic intestinal catarrh (see page 190), or of intestinal stenosis (see page 250). Closure of a narrow portion of the intestines is not at all rare if the bodies have been moved to such places by peristalsis and have there become wedged in; the closure may then be due to the foreign body alone or to it and the masses of feces which accumulate at this site.

The site of the obturation varies according to the derivation of the obturating body. In horses genuine calculi and concrements, also lumps of feces generally become wedged into the first portion of the small colon, more rarely somewhat more towards the anus, occasionally also into the pelvic flexure of the large colon; desiccated feces (chaff) are also sometimes found in the last portion of the ileum or of the duodenum which they obturate rather suddenly (see page 222). The large intestine is usually obturated by fecal agglomerations in dogs. In all other cases (foreign bodies, intestinal parasites) obturation usually occurs in the horse and in all other animals, in the small intestines.

If the obturating body does not stretch the part closed up, because the comparatively large mass is simply held back by the narrowing portion of the bowel without interfering with it, as is, for instance, the case with large calculi situated in the stomach-like dilatation of the colon, then the obturation of the intestinal lumen leads to the same consequences as they are found in fecal impaction (see page 222). If, however, the foreign body has been pushed into a narrower portion of the intestine, it will stretch and irritate the intestinal wall and cause attacks of powerful convulsive contractions, that is, colicky pains. In such and also in the previously mentioned cases, as already explained when discussing the pathogenesis of fecal impaction, convulsive painfnl contractions of intestinal portions situated nearer to the stomach will occur likewise. If the body

wedged in the intestinal lumen is pointed or sharp-edged, it may produce continnons pain.

As in all forms of obturation of the intestinal lumen, the peristalsis becomes abolished behind the obturated point, and often increased for some time in front of the obstruction; later, however, the peristalsis also ceases in the portion between the obstruction and the stomach as stretching of the bowel occurs from the accumulating contents. Bloating occurs only after some time in these portions, if their contents are more or less fluid; the meteorism does not, however, reach a very high degree, because normal intestinal contents do not form much gas and the latter is easily absorbed. In closure of the posterior portions of the large bowel feces collect in front of the obturated point if the appetite has in the mean time remained fairly good; the feces desiccate and extend the lumen of the bowels. Secondary gastric dilatation does not infrequently occur under such circumstances in horses.

Pressure or direct injury exerted by the obturating body not infrequently brings on necrosis or inflammation, and these may extend to the internal layers of the wall, even to the peritoneal covering. Necrosis or convulsive contractions of the intestinal wall may not rarely cause intestinal rupture. As long as complications (inflammation, rupture, possibly high degrees of bloating, dilatation of the stomach) are absent, there are no general symptoms, such as are generally seen after absorption of bacteria and their toxins, after peritonitis and in consequence of severe bloating.

Symptoms. In **horses** the visible signs and symptoms of internal obturation are identical with those of impaction in a part of the cases, particularly if the first portion of the small colon has been closed up (see page 223), except as to defecation and as to the onset of the affection. In obturation the clinical picture develops suddenly in distinction from impaction of feces, and after one or two defecations, complete constipation comes on. In other cases there are severe attacks of colic without any premonitory symptoms which recur from time to time, usually at shorter intervals. The animals throw themselves down recklessly and roll energetically; exceptionally, when the obstructing concrements have sharp borders, the animals do not throw themselves or roll, because this only increases the pain due to injury by the calculi. The animals betray pain by restless tripping, by looking around towards the abdomeu, shaking the head, and occasionally by painful neighing (authors' observation). Abnormal positions, such as dog fashion, kneeling postnres, peculiar stretching, may also be frequently observed. They are not of any particular significance, because they are caused by the abdominal tension or by the pressure upon the diaphragm and are frequently seen in the course of other affections.

Directly after the onset of the first symptoms, defecation

occurs only a few more times, often only once or twice, then no more feces or intestinal gases are voided, in spite of the fact that the animals make efforts at defecation. If the obturation takes place gradually, diminished defecation occurs for some time, and if the concrements which form the impediment are quite irregular the discharge of thin-fluid feces may be kept up, because these can pass between the irregular surface of the calculus and the intestinal wall, through the clefts which have remained open (authors' observation).

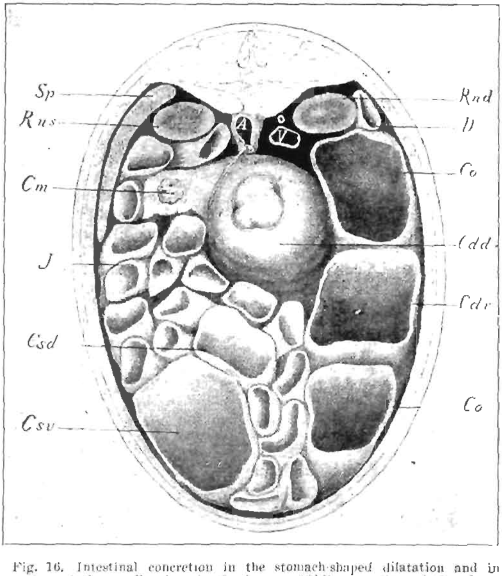


Fig. 16. Intestinal concretion in the stomach-shaped dilatation and in the first portion of the small colon, in the horse. (Oblique section of the abdominal cavity, above through the 1st lumbar vertebra, below through the region of the *Sp.* spleen; *Rns.* left, *Rnd.* right kidney; *Cm* intestinal concretion in the first portion of the small colon; *J* loops of the jejunum; *Csd* left upper colon; *Csv* left lower colon; *D* duodenum; *Co* cecum; *Cdd* stomach-shaped dilatation of the colon with an intestinal concretion; *Cdv* right lower colon; *A* aorta with the Art. mesent. ant.; *V* posterior vena cava.

Rectal examination shows that the posterior portion of the rectum is entirely open. If the arm is introduced up to the shoulder, the examiner may feel the obstructing calculus or the concrement, in horses which are not too large, in the first portion

of the small colon, immediately in front of the anterior pole of the left kidney; possibly also in the median line. Obturation in the pelvic flexure or in the small colon (socalled abdominal portion of the rectum) can be felt with ease in all horses. An obturation of the ileum or of the dilatation of the large colon can likewise be felt, unless the patients (horses) are very large (see Fig. 16). One can rarely expect to find masses of ascarides in the small intestines.

Calculi and concrements may usually be felt on palpation as hard, unyielding, frequently uneven, nodular bodies, but their true shape may be covered up by fecal masses which have become deposited on them. The intestinal wall at the site of the obstruction is usually moderately tender, but it may be extremely tender, as the authors have seen in one case, so that the patient rears and throws himself down whenever the hand approaches this place. The obturating body may exceptionally be in the rectum, so that it can be felt directly by the introduced hand.

The temperature is at first normal, and respiration and pulse deviate only very inconsiderably from the normal figures in consequence of restlessness; the pulse remains usually below 50 in the beginning. Obturation of the anterior portions of the small intestine forms an exception from the general rule because the early occurrence of dilatation of the stomach or of enteritis frequently raises the pulse rate shortly after the onset of colicky symptoms. Dilatation of the stomach may cause belching, retching or vomiting.

Obturation due to retention of the meconium on the second day after birth, causes marked restlessness, pawing with the front legs, wagging of the tail, stretching, pressing, lying on the back, dog-fashion squatting, sometimes even convulsions (Töpper).

If there is no improvement one usually sees in the further course, rarely on the first, more generally on subsequent days, an elevation of temperature; also acceleration and weakness of the pulse. These symptoms increase very slowly; they are caused by the onset of complications (enteritis, peritonitis, meteorism, rupture). Rupture of the intestines is not followed by collapse, provided it has been small and, provided only solid feces have gotten into the abdominal cavity, because then bacteria and their toxins are not at once absorbed in larger amounts; but there develops the clinical picture of general acute peritonitis, characterized by unevenness and tenderness of the peritoneum, which can be ascertained on rectal examination; one may also frequently be able to feel free particles of feces in the abdominal cavity. If an exploratory puncture is made, one obtains a fluid exudate containing particles of feces and numerous bacteria.

The clinical picture in **cattle** is identical with that of intes-

tinal stricture (see page 252) (Holterbach), or one observes more or less marked symptoms of colic, obstinate constipation in spite of repeated efforts. The appetite is poor, rumination has ceased and moderate bloating usually occurs. Intestinal sounds are absent. Rectal exploration sometimes reveals the presence of an obturating foreign body in the intestines, which may be felt in the right half of the abdominal cavity (Spörer, Feser).

In **dogs** we observe obstinate vomiting, possibly colic, absence of appetite, but increased thirst and complete constipation. The animals are less lively, hide themselves, whine and cry on getting up, on moving, and also while at rest; they often change their place of rest, are cross and excitable. In the further course of the disease they become increasingly apathic; there may be convulsions, elevation of temperature, acceleration of pulse, rapid emaciation. Palpation of the abdomen reveals marked tenderness in places, and possibly the presence of the obturating body (the latter may often be easily detected during narcosis). A pointed foreign body (tooth pick) gave rise, after perforation of the intestines, to the formation of an abscess. In a dog with infrapapillary obturation of the duodenum, Dasch observed motor insufficiency of the stomach with tetany which disappeared after the operative removal of the foreign body (see page 155).

In a pig in whose colon a piece of wood had become lodged without occluding the intestinal lumen, Otto did not observe any symptoms of disease.

Course. The disease is usually of short duration in larger animals and in complete obturation; it then ordinarily extends over two to three days, rarely over a longer period; it may, however, occasionally last longer, one to two weeks (authors' observation) or even more. (In a case reported by Felder, the horse died after a sickness of 30 days). In dogs, on the contrary, intestinal obturation often lasts from one to three weeks. Complete obturation is often preceded by the symptoms of intestinal stenosis with occasional, more or less severe, disturbances of defecation or colic.

Recovery occurs rarely spontaneously or only upon internal treatment. It may, however, occur if the obturating body is not too large and can therefore be pressed towards the anus and finally expelled by convulsive contractions of the intestinal wall or exceptionally, if the foreign body is pointed, penetrate the abdominal wall (observed by Navez and Renard in a horse). Only very exceptionally a calculus returns from the beginning of the small colon into the stomachlike dilatation. In Atzinger's case in a horse with light impaction colic a tablespoon 22 cm. long was passed, whereupon the horse recovered.

In the majority of cases complications lead to a fatal issue, the end being ushered in by febrile temperature, frequent and

small pulse, collapse; the intense abdominal pains frequently cease suddenly (rupture) or gradually before death occurs.

Diagnosis. Obturation of the intestines can be diagnosticated *positively only after the obstructing body has been felt,* either by rectal exploration or, in smaller animals, through the abdominal wall. By means of X-rays one may often detect the foreign body in dogs. The history may furnish valuable data in dogs, and in horses a history of continued feeding with bran and crushed grain or of a long stay on sandy, marshy pastures, creates the suspicion of obturation by calculi or concrements

The disease is distinguished from primary fecal impaction by the fact that a part of the cases (of obturation) is complicated by severe abdominal pains; that a distension of the rectum by fecal masses is absent in the beginning and eventually even later, and that there is obstinate constipation from the start. Fecal accumulation in the rectum is not seen in intestinal displacements or in thrombosis of mesenteric vessels, but severe bloating of some portions of the intestines and a weak accelerated pulse are observed early.

Treatment. In the **horse** the expulsion of small calculi may be brought about by laxatives, such as castor oil (500-600 gm. with olive oil, with ether 50-70 gm., or with mucilaginous substances, some radix liquiritiæ as an electuary), also pilocarpine (0.20-0.30 gm.), arecolin (0.05 to 0.08 gm.), or cesol. Thus Schaidler observed, following 2 injections of eserin-arecolin and the introduction of water for several hours, the passage of a concrement the size of a child's head, the nucleus of which was a calculus the size of a child's fist. Because of danger of rupture of the intestines, eserin or barium chloride should not be given alone. Calculi that have become lodged near the anus may be removed manually with rotating movements or after having first been broken up with the hand (successfully in a case reported by Kuerschner).

One may also cautiously attempt the breaking up of a concrement lodged in the first portion of the small colon. If the calculus is situated more anteriorly one might try to push it into *the stomachlike dilatation (Colin); this attempt is,* however, rarely successful on account of the accumulation of feces in front of the place of obturation, as the author's observations in this respect have shown. One may also try the injection of large amounts of water (30-40 liters at one time).

Töpper recommends Masch's meconotorium for the removal of meconium which is impacted in the rectum; this instrument has the shape of a spoon and it must be introduced repeatedly with care. After cleaning out the rectum an injection is given (1 quart of 1% lysol solution) and internally 1.0 calomel with 50 gm. castor oil. In order to remove feed balls from the intestinal tract of colts, Bellec introduces water into the rectum in a peculiar manner, after first *having removed any accumulated gases by puncture of the intestines.* The animal is cast and a tobacco-pouch suture placed around the anus. A rubber tube 10 to 20 cm. long and 18 to 25 mm. thick is introduced into the rectum and the anus is drawn tightly around it by means of the suture. According to

the age of the animal, 8 to 10 liters of lukewarm water is introduced through the rubber tube into the rectum from a container elevated 1.5 meters. In spite of the restlessness and straining of the animal, he waits 3 hours; he then removes the rubber tube and the suture and injects eserin under the skin. Not infrequently a repetition of the treatment is necessary, in which case the animal is placed on half ration for several days.

If these attempts fail or if the size and shape of the obturating body exclude the possibility of success from the start, laxatives should not be given, but a laparo-enterotomy might be tried. However, the latter will be successful only in very exceptional cases, since the union of the stomachlike dilatation of the large colon into the small colon is usually displaced into the latter, and it therefore becomes impossible to draw this portion of the intestine into the wound of the abdominal wall, because the former is too firmly connected with the upper abdominal wall. (Hobday succeeded in removing the intestinal calculi in only 1 out of 7 laparotomized horses, and even this animal died at the end of 2 weeks; in 1 case of Richard, Dollar and Rogers, in 1 case each of Hall, Roeder and Lowe, in 3 cases of Marek and Plosz, the horses died; 1 case of Felizet recovered after the removal of a calculus the size of a child's head). Accordingly the operation seems indicated only when the small colon with its long mesentery or the pelvic flexure is obstructed. Ghisleni succeeded in this manner after 2 operations in successfully removing an intestinal calculus from the cecum of a horse in spite of the fact that the cecum had formed many adhesions. Hoffmann recommends the removal of the calculi wedged in the posterior portion of the rectum, by introducing the hand through the laparotomy wound and pushing carefully the stone towards the rectum where it will be grasped and extracted by the hand of an assistant. Large calculi can, of course, not be removed by this route.

Obturating foreign bodies in **cattle** may also be removed by operative procedure, as is shown by the observations of Spörer and Feser.

The expulsion of foreign bodies wedged in the intestinal tract is favored in **dogs** by the administration of laxatives. Foreign bodies in the rectum can be removed by hand or with proper extracting forceps. In Jakob's case in a dog a piece of rope 6.5 meters long was removed piecemeal partly by vomiting and evacuation, but partly only after the administration of laxatives and abdominal massages. If these methods are not successful, or if they are not applicable from the start on account of the nature of the obstructing body, laparoenterotomy may be successfully performed, as is shown by the reports of Felizet, Siedamgrotzky, Fröhner, Degive, Plósz, Marey and others. It is, of course, not advisable to wait long with an operation if it is at all indicated.

Prophylaxis. This may require, according to the prevailing conditions, change of feed, addition of dry roughage (hay,

straw) to the green clover shortly before blossom time or to rushes, increase in ration, addition of calcium preparations to the feed, or removal of intestinal parasites. Stenosis of the intestines is a designation for a group of diseased conditions differing in nature, all of which, as a rule, may result in a gradual decrease in the caliber of the intestinal lumen.

22. Narrowing of the Intestine. Stenosis intestini.

Stenosis of the intestines is a more or less chronic disease with a generally progressive diminution of the lumen of the intestine at a circumscribed place.

Occurrence. Stenosis of the intestine is a rare affection. It occurs in horses with about the same frequency as obturation of the intestines (see page 239).

Etiology. The following are the pathologic conditions which may be the causes of intestinal stenosis: Scar formation in the intestinal wall caused by injury through foreign bodies, by ulceration, possibly after gangrene of an intussuscepted portion of the bowel, produces narrowing of the intestinal wall (strictura intestini) in consequence of cicatricial contraction of the newly formed connective tissue. Upon this basis intestinal stricture is usually found in the small intestine, the small colon, or the rectum, in domestic animals with the exception of the hog. According to Bruckmüller, Sikorski, Bolton, in horses occasionally, but according to the reports of the Berlin pathological institute frequently, a cicatricial stenosis of the ileocecal opening is noted, due to chronic inflammation of the mucosa and caused by the continuous irritation of coarse feed (short-cut chaff, buckwheat straw). Ligation of the prolapsed rectum often causes stenosis in hogs (Johne).

According to Petit and Germain and Brieg, the stenosis in the ileum of the horse resulting in hyperplasia and thickening of the muscular layer is probably due to a continual safety-valve-like operation of Bauhin's valve when large amounts of fecal matter and gases have accumulated in the large intestines, and possibly also to a compression of the cecum and ileocecal valve. Thus pronounced stenosis does not often occur, but lighter forms are more frequent. Ligation of the prolapsed rectum in hogs leads to stenosis (Johne). Bianchi found as the cause of finally fatal stenosis of the ileum in an ox a layer of lime about 1 cm. thick deposited between the mucosa and the muscularis forming pronounced longitudinal folds. Congenital stenosis of the duodenum of the dog was observed by Schreck.

Chronic peritonitis which produces newly formed and subsequently shrinking cicatricial connective tissue on the serosa

of portions of the intestines, may cause stenosis of the intestines of variable extent. (Ross, Zürn, Dignac, v. Ow., authors' observation.) Intestinal stenosis is frequently produced in chronic adhesive peritonitis, in consequence of kinking in loops of intestines which have become adherent to the abdominal wall or to neighboring organs, because the two branches of the adherent loop pull upon the place of attachment by their own weight. (Avérous, authors' observation.) Sometimes localized subacute peritonitis may produce intestinal stenosis in this manner, as has been seen in the horse (authors' observation).

Neoplasms in the intestinal wall are not infrequently the cause of intestinal stenosis (Achilles found intestinal tumors, mostly sarcomata in 0.2% of the horses slaughtered in the stock yards at Leipzig). Most frequent are polyps of the mucosa; they may be multiple in this place. Sarcomata are usually found in the small intestine; they are either firm or soft, and they also assume an infiltrating form (Rabe, Kitt) with the histologic structure of a lymphosarcoma. Melanomata occur in the cecum and rectum of the horse (Harrison, Csokor). Carcinomata are frequently seen in the region of the anus of the dog (Fröhner); they are, however, rare in other parts of the intestinal tract and in other animals. (According to the compilation of Achilles only six cases have so far been reported in the horse); these carcinomata in horses have no tendency to undergo ulcerative changes. As rare neoplasms must be mentioned: fibromata, myxomata, lipomata, leiomyomata, adenomata, adeno-carcinomata, actinomycomata (the latter are of course, not true tumors). (Markus observed stenosis of the rectum, and Favero stenosis of the ileum due to actinomycomata of the intestinal wall.) Here we may also mention lymphadenosis of the intestinal wall, which, when decided thickening of the mucosa and submucosa takes place (2.5 cm.) and the intestine is converted into a stiff walled tube, may produce symptoms of stenosis of the intestines.

Guénon removed from the rectum of a horse a pediculated, very hard tumor the size of a walnut; it was, according to Petit, formed in such a manner that a calculus had formed in a gland of Lieberkühn. This calculus had gradually distended the mucosa and had drawn it out with the formation of a pedicle. In a case of Cadéac congenital valve formation in the rectum had been the cause of stenosis.

Calculi, balls of feces, feed and hair balls, conglobates, foreign bodies, not infrequently cause stenosis before entirely obstructing the lumen of the intestines. Circumscribed dilatations of the intestinal wall (diverticulum intestini), which may either be congenital (diverticulum of Meckel in the ileum) or acquired through the action of heavy bodies (sand, calculi, fecal balls), and may constrict the lumen of the part where they are located by compressing it when filled or by twisting it through traction. These forms of diverticula are most commonly found in the horse in the last portion of the ileum, in the large intestine, or in the small colon. Joest found two true diverticula

lying opposite each other, in the small colon of a horse which had had periodical attacks of colic owing to expansion of the diverticula which did not affect the lumen of the intestine otherwise. In Neuerschwander's case a diverticulum of the cecum had grown to the testicle and was carried along with the latter when it descended into the inguinal canal. When the animal was castrated the diverticulum was cut open, thus resulting in an intestinal fistula. In a case reported by Mantsch a diverticulum was formed on the small colon by traction of the hematome in the intestinal wall.

Compression of the intestine by an abscess or a neoplasm formed in the mesentery or by enlarged abdominal organs (gravid uterus, filled rumen, enlarged ovary, enlarged prostate); (enlarged spleen due to hemorrhage in birds, exceptionally to an enlarged bursa, as the result of a collection of exudate or hyperplasia of its wall [Schantyr and Paukul]) may not infrequently cause stenosis of the bowels. Hendrickx saw in a horse a compression of the intestines due to a detached, partly ossified cryptorchitic testicle, which subsequently broke through the atrophic intestinal wall into its lumen. In Engler's case the rectum in a hog was compressed markedly by the pelvic bones which were rachitic and deformed. In Fleischhauer's case in a mare the rectum was compressed by a hematome forming between the small colon and the ligamentum latum uteri during parturition, while in a case observed by Petit and Houdemer in a mare a teratoma probably developing subperitoneally caused a fatal compression of the large intestines. A clinical picture corresponding to stenosis of the rectum is often observed in cattle, according to Stalfors, due to wounds in the rectum produced either wilfully or by the application of enemas or by the careless use of the thermometer. Interference occurs not so much as the result of subsequent inflammatory swelling of the intestinal wall as of reflex inhibition of contraction of the intestines lying anteriorly to the wound.

Intestinal stenosis may also be caused by **internal herniae** before they become strangulated; the clinical picture then usually terminates by the symptoms of complete incarceration. **Parasites** (ascaris, gastrophilus larvæ [Kater, Rexilius], intertwined *tæniæ) will only exceptionally produce a picture similar to intestinal stenosis.*

Pathogenesis. According to the character of the enumerated causative factors, the intestinal lumen usually becomes narrowed very gradually; in exceptional cases the stenosis remains stationary or it only occurs temporarily (diverticulum, internal hernia, compression caused by an overfilled rumen). **In all cases interference with the movement of intestinal contents by the stenosis takes place earlier, more pronouncedly and more *frequently, the smaller the respective portion of the***

intestines, the firmer the feces in the same, and the more abundant and drier the ingested food. The fecal masses accumulating in front of the narrowed point stimulate the intestines to forcible contractions at the place where the accumulation has taken place; some of these are convulsive and cause colicky pains. These forcible contractions will finally succeed in pressing the accumulated feces through the stenosed portion and the time which elapses before this occurs depends upon the degree of stenosis and upon the consistency of the feces. After the feces have passed the narrowed point, the convulsive contractions and the pains cease. Hypertrophy of the forcibly contracting muscularis and dilatation of the intestine will develop in the course of time. The nearer to the stomach the constriction is situated, the sooner after the ingestion of food will abdominal pains come on, and a chronic dilatation of the stomach will usually develop; such a condition may, however, also develop in stenosis of the large intestine. In the latter condition attacks of pain occur independently of the ingestion of food; in stenosis of the rectum they commonly come on before defecation. Those portions of the intestines which are situated between the stricture and the anus do not contain a great deal of feces, hence they act sluggishly. The feces accumulate and decompose in front of the stricture and frequently become instrumental in producing chronic intestinal catarrh, which influences the nutrition of the animals unfavorably. The stenosis may finally lead to complete occlusion of the bowels.

Symptoms. In the **horse** and in **cattle** a clinical picture is observed at variable intervals, which is identical with that met with in impaction (see pages 223 and 225); the attacks may last for a few hours only, or for several days (socalled habitual, recurring or periodic colic). With remitting mild or on the contrary, very severe attacks of colic, there develops afterwards an incomplete constipation. In stricture of the small intestine the attacks usually occur shortly after the ingestion of food, and they are often followed by dilatation of the stomach (see page 140). In stenosis of the rectum restlessness is noticed before defecation. In cattle there occur, aside from the symptoms of intestinal obstipation, those of a periodically recurring atony of the stomach or overfilling of the rumen (Storch).

During the attacks of colic, pulse, respiration and temperature remain, as a rule, normal or are only changed insignificantly; an exception to this rule is noted, however, when a dilatation of the stomach or enteritis has set in, or where the underlying cause of the stenosis of itself leads to fever (abscess, peritonitis).

During the intervals between the attacks the animals either appear perfectly healthy, as for instance in moderate stenosis of the small intestine, or defecation is scanty, occurring only after longer intervals, or instead of being dry and small, the

feces may be softer than normal and has a foul odor, a condition which is due to secondary intestinal catarrh.

Rectal examination often does not merely reveal the seat of the stenosis but also its cause. In stenosis of the rectum the hand meets an impediment, either near the anus or somewhat more distant from it, which cannot be removed either by spreading the finger or by the injection of lukewarm water (this distinguishes true stenosis from temporary constriction, which can be overcome by the means indicated); one often also can feel feces with the finger pushed through the constriction, sometimes a dilatation and occasionally a kink. In the course of the examination one should look for wounds in the wall of the rectum, which can sometimes be felt in cattle as an insignificant induration with a central opening. At the same time the animal will evince pain and traces of blood will be found adhering to the palpating finger. If the arm is introduced up to the shoulder, narrowing of the small colon may be detected, because all loops of this part of the bowel are in a portion of the abdomen which can be reached from the rectum. A stricture in the posterior third or possibly the posterior half of the left loop of the large colon, or in the upper half of the cecum, the terminal portion of the ileum, or in the loops of small intestines situated below the renal region, may be detected in the same manner, and in horses which are not too large they may even be found in the stomach-like dilatation of the colon. One finds the intestinal wall thinner at the constricted portion, possibly adherent to a neighboring organ, or one may detect a large abnormal formation in its lumen or in the neighborhood. In front of the stenosis there is usually an accumulation of feces. Palpation of the right half of the abdomen in cattle may show stenosis of the rectum, stenosis or adhesions of the cecum, stenosis of other portions of the large intestines or of some loops of small intestine.

The presence of a non-incareerated hernia of the diaphragm may be assumed with some degree of certainty from the presence of marked loud intestinal sounds in the thorax, of tympanitic sounds, varying at short intervals in intensity and pitch, and heard at the posterior portions of the thorax; also from the occurrence of intense dyspnea in walking down steep roads or from lying on the side. Diaphragmatic hernia may also lead to asthmatic symptoms (Bärner). Loud intestinal sounds are also heard over the thorax whenever peristalsis is much intensified and a tympanitic sound over the posterior lower parts of the chest can be heard not infrequently if gas containing portions of the large colon have for some reason or other been pushed towards the thorax and have pressed the diaphragm into it.

Intestinal stenosis in **hogs** and **carnivora** is accompanied by gradually increasing obstipation, abnormally thin strings of feces are evacuated, or very small lumps of feces if the stenosis

involves the anal portions of the colon or rectum. Rectal exploration reveals a constriction of the rectum somewhere in the pelvis (tumors, enlarged prostate, enlarged uterus, cicatrix). Palpation of the abdomen shows a firm, hard body in the intestines, or in the mesentery, diffuse adhesions with the formation of lumps or the enlargement of certain abdominal organs.

Course. Stricture of the intestines does not lead to any disturbances for some time; after a variable interval of time, however, there occur, in larger animals, particularly in horses, attacks of abdominal pains of variable duration. They recur at first after long intervals, sometimes only after one to two years, but they become more frequent as the disease progresses. The intervals depend to a certain extent upon the nature of the feed and upon the amount of work required of the animals (Bärner). As the attacks become more and more frequent and as obstipation increases continually, complete and permanent intestinal obturation may occur. In the mean time the animal has become emaciated and death occurs from enteritis, peritonitis, rupture or displacement of the intestines, exhaustion. In some cases an animal in a fair state of nutrition may succumb, during one of the first attacks, to rupture of the stomach or intestines, or to enteritis. Spontaneous healing will sometimes take place, as in the case of Pante, following the evacuation of an abscess which had formed along the cecum into the lumen of the intestines, and somewhat more frequently in stenosis of the rectum due to wounds.

Diagnosis. Stenosis of the intestines and its origin can only be positively diagnosticated by rectal exploration or by palpation of the abdomen. In the absence of positive findings the presence of stricture of the intestine may be assumed with a certain degree of probability from the history of certain preliminary diseases, leading to narrowing of the lumen of the bowel, and leading in shorter and shorter intervals to attacks of colic with constipation, especially after the ingestion of dry or firm feed; at the same time general symptoms are absent, and a gradually increasing obstinate obstipation develops in smaller animals. However, the symptoms here enumerated do not permit the exclusion of fecal impaction or thrombosis of intestinal vessels from other causes.

Treatment. In the majority of cases the treatment recommended against fecal impaction is indicated (see there). In the cases of narrowing of the intestine, which are due to a nonincarcerated hernia or to an excessively large foreign body, it is not advisable to use laxatives with intense action, because incarceration of the hernia or complete occlusion by the foreign body may occur. In such cases one should confine oneself to the use of the neutral salts. In rectal stenosis, local treatment

may be assisted by mild laxatives. If dilatation of the stomach has occurred the use of the stomach tube and lavage of the stomach are indicated.

If these methods are not successful, or if an improvement cannot be expected from them in consequence of the nature of the stenosis, relief may be attempted by operative procedures.

Pediculated tumors of the rectal wall should be removed by crushing or ligation of the pedicle; tumors with a broad basis or those of the paraproctal connective tissue must be removed by excision. (Dornis removed a fibrosarcoma weighing 4,450 gms. from the wall of the rectum of a horse), or possibly by the injection of drugs hastening resorption of the tumor (Leicht saw the disappearance of a tumor as large as a man's fist and located 24 cm. anterior to the anus in a horse, after a single injection of 6 cm. of a 6% Jodvasoliment). Periproctal abscesses must be properly treated after an incision. Douville removed a cyst from the rectum of a horse by puncture, followed by injection of Lugol's iodine-iodide of potash solution. Certain forms of stenosis situated in front of the pelvis may be attacked through the rectum (tearing of adhesive bands, displacement of enlarged organs). (Guittard temporarily relieved in this manner a stenosis caused by an enlarged rumen.) Stenosis of the rectum due to wounds can be successfully treated, according to Stalfors, by the application of enemas with mucilagenous drugs. When severe straining is present, cocain and chloral hydrate are indicated.

The great majority of cases of stenosis formed in parts that are distant from the pelvis can be treated only by a laparotomy or eventually an enterotomy. It is absolutely necessary first, to ascertain the exact nature and seat of the stricture by rectal exploration, because laparotomy is a dangerous operation, particularly in the horse, and its successful performance requires the selection of the proper place for the incision; the latter should, if possible, be made at a place nearest to the site of the intestinal stricture. In smaller animals, however, the best place for the laparotomy incision is, as a rule, the median line (linea alba). Stenosis caused by adhesive bands can be removed most easily, because all that is generally necessary after laparotomy is the severing of the constricting bands. Plósz & Marek have successfully performed laparotomy in such cases. Deghilage broke up adhesions of the small colon with the hand introduced through an incision in the vagina. De Meis & Parascandolo have removed a carcinoma of the cecum in a dog by laparo-enterotomy.

Proper regulation of diet (green feed, bran slop, thin flour pastes, milk, occasionally molasses) usually postpones the development of complications and of an unfavorable termination.

23. Obstruction of the Mesenteric Arteries. Thrombosis et embolia arteriarum mesenterialium.

(*Verstopfung der Gekrösarterien; Thrombotisch-embolische Kolik* [German]; *Congestion intestinale* [French].)

Thrombosis of the mesenteric vessels is due, as a rule, to inflammation and obstruction, caused by sclerostoma larvæ; it occurs in this connection only in horses, and leads often to frequent temporary attacks of colic.

Many French authors classify intestinal disturbances due to thrombosis and embolism with "Congestion intestinale," to which also belong catarrhal and inflammatory conditions (see page 198). This cannot be approved, since it throws very heterologous affections together under one head.

Historical. Larvæ of sclerostomum were first seen in the mesenteric arteries by Ruysch in 1665 and similar observations were then made by several other authors. The causal connection between these parasites and attacks of colic has since the beginning of the last century been pointed out by several authors, especially by Rigot, Schutt, Hering, Reynal, Bruckmüller, Prehr and others. Bollinger (1870) made systematic examinations of this pathologic condition. Modern investigations of verminous aneurysm of the horse have more recently been made by Sticker (1901), Glage (1905), Adelmann (1908). Disturbances of the circulation of the blood and of the functions of the intestines following thrombosis of the mesenteric arteries have been studied by Marek (1907).

Occurrence. Bollinger's investigations, which have been confirmed by other authors, have shown that 90 to 94% of all horses with the exception of foals have a verminous aneurysm. However, the statement of this author must be considered as entirely unjustified, that one-half of all fatal cases of colic are caused by thrombosis or aneurysm and that all cases in which colic appears suddenly and without any apparent cause are due to this condition. The opinion of Stietenroth, according to which 80 out of every 100 cases of colic terminating fatally must be ascribed to **Sclerostomum**, is likewise unjustified. More recent observations (Matthiesen, Franke, Schultze, Pöppel, Glage, Folmer, Bang, Jensen). More recent observations (Matthiesen, Franke, Schultze, Pöppel, Glage) prove that the disease also occurs in foals from the third month on. Pöppel, indeed, has seen, in a foal ten days old, a dilatation of the anterior mesenteric artery as large as a pigeon's egg, and in it a thrombus containing larvæ of sclerostomum.

Verminous aneurysm appears to be more common in foals than is usually assumed. Among thirty-five slaughtered foals, from five to several months old, Glage found aneurysm of the anterior mesentery in fifteen, varying in size from a walnut to a fist. Folmer also reports the same percentage. Bang and Jensen frequently observed fatal cases of verminous aneurysm in young colts.

The following figures about the frequency of thrombotic infarction of the intestinal wall are given from various sources: Dresden Clinic, 1899-1908, in horses dead after attacks of colic, 9.5%; Berlin Clinic, 1891-1907, in 3%; Prussian army, 1892-1908, 6.6%; Budapest Clinic, 1900-1909, 14.4%. Wall claims that thrombotic-embolic disturbances of the intestines are quite rare. Indeed there seem to be great differences in the frequency of the affection, according to periods of time and to various countries.

A fairly correct conception of the clinical significance of thrombosis of the mesenteric arteries in grown horses can be obtained from the reports on the horses in the Prussian army, according to which there is an average of 3 fatal cases of thrombotic-embolic colic a year per 10,000 horses. If we should place the number of nonfatal cases at the excessively high figure of 5 times this number, then there would be 18 for every 10,000 horses—that is, 1.8 cases of colic due to thrombosis of the mesenteric arteries per 1,000 horses, in spite of the fact that we find these lesions in 90% to 94% of all grown horses.

Etiology. According to the investigations of Sticker, thrombosis of the mesenteric arteries of the horse is caused by the **larvae of sclerostomum vulgare** Looss (Scl. bidentatum Sticker) which are taken up with the drinking water, by gnawing at the stable wall or other objects contaminated with manure, also on wet and swampy pastures. For particulars, and also for the development of sclerostoma see the chapter on palisade worms in the intestine.)

In horses as well as in other animals there occurs exceptionally a transportation of emboli into the mesenteric arteries in consequence of cardiac disease, endocarditis and aneurysm of the thoracic aorta. The emboli in the mesenteric arteries are usually not followed by serious consequences on account of their small size. A thrombus from the posterior aorta may also become directly lodged in the mesenteric arteries (Dornis). The authors have seen the case of a dog where an ulcerative endocarditis was followed by an embolic closure of arteries of the small intestines, and this led to hemorrhagic infarct of the intestinal wall. A. Koch and Leibenger have each seen a case of aneurysm of the anterior and posterior mesenteric artery in the hog.

Pathogenesis. The larvæ of sclerostoma, according to general belief, adhere to the intima and even bore into it, after they have gotten into the mesenteric artery; this causes inflammation and the formation of blood coagula, also after some time, a thickening and a dilatation of the vessel wall. Sticker holds a different view, namely, that the larvæ become lodged in the vasa vasorum, cause infarction in the media of the vessel, and then mesoarteritis, or peri- and endarteritis. These changes are seen most frequently in the anterior mesenteric artery, or in the art. ileo-cœcocolica, rarely simultaneously or exclusively in a branch of this artery; also in the posterior mesenteric artery or in the art. cœliaca.

In 108 cases of aneurysms in horses Hering found the art. mesent. involved 100 times (92.6%), the arteria cœliaca five times (4.6%), the art. mesent. post. twice

(1.8%) and the art. renalis once (0.9%). Bollinger's sixty cases were distributed as follows: Fifty-three times (83.3%), the art. mesent. thirty-two times in the main trunk; ten times each in the upper and in the lower branch; once in a branch of the small intestine; twice (3.3%) in the abdominal aorta; twice (3.3%) in the art. cœliaca; once (1.6%) in the art. mesent. post.; and twice (3.3%) in the art. renalis. In twenty-nine cases of aneurysma in the art. mesent. ant., Wall saw the aneurysm in the main trunk of this artery only once (3.4%); in the other twenty-eight cases (96.6%) it was found in the art. ileo-cœcocolica. Adelmann found in all of his ninety-five cases the aneurysm in the art. mesent. ant.; in seventy-three horses this artery and its branches alone were involved; in one horse the art. mesent. post. was affected simultaneously; in four horses the posterior aorta; in two horses the art. cœliaca; in one horse the portal vein; in one horse the splenic artery and in three horses the pulmonary arteries. In these eighty-five cases thrombosis was usually seen in the art. ileo-cœcocolica and also sixteen times simultaneously in the art. col. ventralis; ten times in the art. col. dors.; seven times in the art. ileo-cœcalis, and three times in an artery of the small intestine.

The formation of a thrombus leads to a narrowing of the lumen, provided that dilatation is not commensurate with the diminution of caliber, or if the end of the thrombus projects into the lumen of a branch.

Stenosis or complete obstruction of mesenteric arteries may be brought about in a variety of manners. It may occur that a thrombus formed somewhere increases gradually in size until it closes the lumen completely. There is also the possibility that two or more neighboring and anastomosing arteries are affected simultaneously by thrombosis; in such a case even a stenosis of not very high degree may become dangerous. One cannot infrequently see that in non-obturating thrombosis of the art. ileo-cœcocolica or of the art. colica ventr., blood can get in sufficient amount into the colon through the arter. colica dorsalis, so that its blood circulation remains fairly normal. If, however, the art. colica dors. becomes stenosed or completely obstructed, local circulatory disturbances will come on without fail. Cases are not rare in which a thrombus formed in a main trunk will become elongated, and will reach and obstruct the opening of an arterial branch. A thrombus formed in the art. ileo-cœcocolica and growing peripherally may reach or even grow into and obstruct the art. colica ventr., or one of the art. cœcales, or the ramus iliacus; if a thrombus in the art. ileo-cœcocolica grows centrally, it may spread into the art. colica dors., or into one of the arteries of the jejunum. In such cases, according to the observation of the authors, obstruction of the branch occurs in the following manner: The blood current moves the free floating end of the thrombus and eventually presses it into the opening of one of the branches of the main trunk. If the terminal free end has once been pressed into an opening, then closure is intensified, because there is a decreased blood pressure peripherally and an increased blood pressure centrally. However, if a sufficient collateral circulation is established, the blood pressure again may become evenly balanced in front of and behind the obstruction, and the thrombotic plug may again become free.

In the larger mesenteric vessels thrombosis may bring

about embolic closure of smaller branches. Since such emboli are, as a rule, small, and since they therefore obstruct only smaller arteries, they do not, as a rule, produce any serious disturbances of circulation, either alone or in connection with the primary thrombus. Only if arranged in arteries one above the other, like the floors of a building, might emboli become dangerous. No doubt every thrombus does not necessarily lead to embolus formation. On the other hand, thrombosis in other

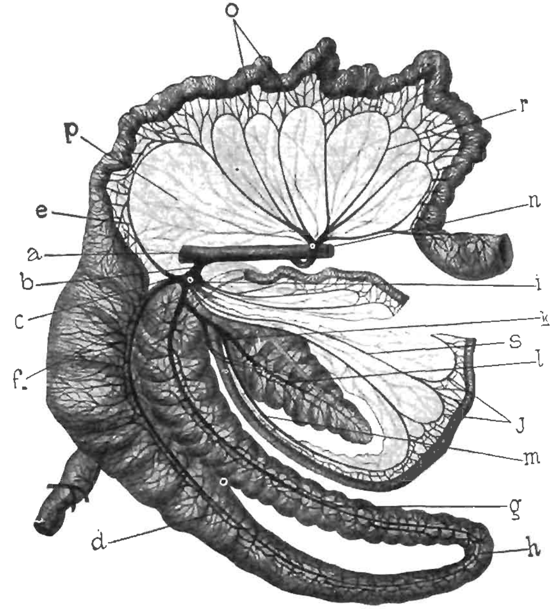


Fig. 17. The mesenteric arteries of the horse. (Adapted partly from Ellenberger and Baum; the vascular anastomoses are drawn after a Teichmann preparation.)

organs or in the aorta, may lead to embolism in the mesenteric arteries.

The effect upon the circulation in the intestines of obstruction of the mesenteric arteries depends mainly upon the size and the number of the obstructed vessels, upon the seat and the degree of stenosis and upon the size of the anastomoses which connect neighboring vessels with those that are thrombosed.

The **arteria mesenterica anterior sive cranialis** (Fig. 17 *b.*) supplies all portions of the intestines with blood, except the oral portion of the duodenum, which is supplied by the art. cœliaca, and the middle and caudal third of the small colon (socalled abdominal portion) of the rectum, which get their blood from the art. mesent. posterior, s. caudalis (*n*). The arteria mesent. ant. forms a short, strong trunk, only a few centimeters long; this gives off, in front and somewhat to the left, the art. colica dorsalis (*d*), which supplies all upper branches of the colon, and the art. col. media (*e*), which supplies the cranial third of the small colon. The two last named arteries usually arise from a common trunk (*c*); somewhat more backward and to the left arise the 17-20 jejunales (*i*), which supply the small intestines. In this manner the main trunk becomes thinner and is now known as the art. ileocæcocolica (*f*); it is two to four cms. long; it first gives off the arter. colica ventralis (*g*), which supplies the lower branches of the colon; the remaining portion, the art. ileo-cæcalis, divides into the art. cæcalis (*l*), lateralis (*k*) and the ramus iliacus (*m*) to supply the cecum and the ileum. The arter. cæcalis lateralis gives off a branch to the lateral wall of the right lower branch of the colon (art. colica lateralis of Franck).

The most cranial artery of the small intestines (art. jejunalis) anastomoses with the arter. pancreatico-duodenalis, that is, with the art. cœliaca in the region of the duodenum, by a tolerably large arterial arch situated in the mesentery. The different arteries of the small intestines communicate with the ramus iliacus by similar archlike anastomoses (*j*). The art. colica ventralis and the ramus iliacus are connected with the vessels of the cecum which among themselves possess rich anastomoses. The art. colica dorsalis is continued immediately into the art. colica ventralis at the pelvic flexure, and this forms the largest anastomosis of the animal body (*h*); the art. colica ventralis also forms anastomoses with the art. col. lateralis. If one adds that there are anastomoses between the art. colica dorsalis and the art. colica media, and also between the latter and the cranial branch of the art. mesent. post. (*e*) and between all branches of the latter (*o*), one can easily see that blood can get abundantly into the territory of the branches of the art. mesent. ant., either from the art. cœliaca or from the art. mesent. post. and into the art. mesent. post., and also from neighboring vessels, even if either one of the arteries named is obliterated and the pressure in it is decreased. It is also important and should be emphasized that intestinal branches, whether they arise from main trunks or from arterial arches, and the branches in the intestinal wall in the mesentery are connected by rich anastomoses, as can be seen in Fig. 17.

Narrowing or closure of the lumen of the mesenteric arteries produces a disturbance in the blood circulation of the intestines only if the arterial pressure in the affected portions of the intestines becomes markedly lower and if consequently the current in the capillaries becomes very much decreased. Marek's animal experiments have shown that total and sudden closure will not be followed by recognizable disturbances of circulation, if an anastomosing arch, situated in the mesentery of the small intestines or of the small colon (Fig. 17 j, o), or a branch arising from these arches or from the arteries of the colon or cecum, has been closed up. The obstruction of an artery of the small intestines (s), which has only two anastomosing arches, or the obstruction in one place of one of the arteries of the colon or with embolism in the peripheral branch. The gradual decrease in the caliber of larger or even numerous smaller arteries will, on the contrary, not necessarily lead directly to severe disturbances, because in this case the anastomosis and the blood vessel which probably is only partly obstructed have had time gradually to become dilated. However, a total occlusion of the trunk of the anterior mesentery artery or of the arteria ileocecocolica, as well as obstruction of both arteries of the large colon or cecum near their origin, and obstruction of several

adjacent arteries of the small intestines, will, even in cases of gradual thrombosis, lead to incurable disturbances of circulation.

Lowering of the arterial blood pressure in the excluded territory causes a decreased velocity of the capillary current, because the difference between arterial and venous pressure, which propels the blood through the capillaries, is diminished. The decrease of the caliber of arteries and capillaries leads to a more or less intense arterial anemia and, in consequence of the retardation of circulation, to an accumulation of carbon-dioxide in the blood and the tissues of the affected territory. The retardation of circulation within a short time produces such nutritive disturbance of the capillary walls that they lose their tonicity and permit the constituents of the blood to extravasate. The loss of tonicity may, however, also be due in part to irritation by the intestinal contents. Capillaries of the intestinal mucosa which have lost their tonicity will become strongly filled by blood from the collateral circulation, and this causes more or less marked reddening of the mucosa, which is followed by hemorrhage.

According to the degree of lowering of the blood pressure, two types of circulatory disturbances in the thrombotic parts may be distinguished; there are, of course, no sharp boundary lines between these two types. In the milder form the occlusion is comparatively small, considering the amount of arterial blood which can still get into the affected territory; the decrease in the velocity of circulation is not very high; hence the extravasation of the blood elements is insignificant and a nutritive disturbance, which can be seen macroscopically does not occur. In the mean time, the arteries supplying the affected territory become dilated, the blood supply soon becomes sufficient, pressure and velocity of current become normal again, generally within a few hours, and the extravasated elements of the blood are absorbed. In the milder form, therefore, there is always a restitution of the blood circulation to normal conditions.

The severe form is characterized by very grave nutritive disturbances of all the tissue elements of the intestinal wall, preferably of the walls of the capillaries and of the mucosa. In consequence of the very considerable retardation of the blood current, the permeability of the capillary walls increases from the second quarter of an hour on, and the blood elements extravasate more and more, in increasing amounts, first into the loose tissue of the mucosa, later also into the other tissues of the intestinal wall, to get finally into the intestinal lumen and into the free abdominal cavity. A backward accumulation of venous blood does not take place. A retardation of the lymph current follows the decrease of pressure in the capillaries and the former produces an accumulation of serous fluid in the lymph spaces of the intestinal wall. All this produces an edematous-hemorrhagic infiltration in the intestinal wall (infarctus

hæmorrhagicus). With the profound disturbance of circulation there develops a necrosis of intestinal villi, beginning at the free end and progressing towards the deeper layers of the mucosa. Necrosis of the mucosa may become manifest after two hours, and the deeper layers show the earliest necrotic changes after twenty-four hours; this indicates that contact with the intestinal contents plays a part in the production of necrosis.

In some few cases of the severe type the circulatory disturbances may disappear, but this, of course, takes a much longer time than in the milder form, sometimes several days. In obstruction of some arteries of the small intestine, a reestablishment of the normal circulation may occur in only the two terminal portions of the bowel, while the hemorrhagic infarct persists in the median portion. The limit up to which the circulation may be reestablished after a sudden arterial occlusion is as follows: Neighboring arteries of the small intestines with five anastomosing arches, occlusion of an artery of the colon or cecum near its origin, provided that their anatomoses or the peripheral portions of these arteries are not likewise thrombosed. The more anastomoses are obstructed or at least partially occluded at the same time, the smaller must be the occluded territory if the collateral circulation may become established at all.

Complete inhibition of the arterial blood supply to some portion of the intestines will cause anemic necrosis. After an artery and its capillaries have become sufficiently empty so that the pressure has been lowered to the pressure of the veins of the affected region, the circulation of the blood ceases entirely and the capillaries contain much less blood than under normal conditions. Since obstruction of all anastomoses of a mesenteric artery, which is a necessary condition for the occurrence of an anemic necrosis, very rarely takes place under natural conditions, anemic necrosis in combination with hemorrhagic infarction will very rarely be seen; such was, however, the case in an observation by Caspar of obstruction in the region of the art. mesent. post. If all larger anastomoses of this artery are obstructed simultaneously or successively, very small anastomoses that remain open will admit enough blood only to produce a hemorrhagic infarction in the two end pieces of the affected intestine, while the median piece does not receive any blood at all. An equilibrium of the blood supply can, of course, not be established in this case.

The function of the intestine is only disturbed in thrombosis of the mesenteric arteries when circulatory disturbances are established; the lack of arterial blood and the proportionately increased contents of the blood and tissues in carbon-dioxide act for some time as a stimulus to peristalsis. Some of the more active intestinal contractions become convulsive, the intestinal tube becomes hard and unyielding, and colicky pains are caused by compression and pulling of terminal nerve filaments in the intestinal wall and its mesentery. Since contractions appear at variable intervals in the excluded portion of intestines, and since their duration is variable, the attacks of pain last a shorter or longer period and appear after variable intervals. If arteries have become thrombosed which supply very large portions of the intestines, we may see a continuous colicky attack, because

individual places in the affected long piece of intestinal tube are necessarily convulsively contracted, so that there may not be any interval free from pain.

In cases with lasting anemia of the affected parts, the erstwhile increased intestinal peristalsis soon becomes sluggish and, as shown by experiments, it ceases entirely after one to two hours. The early cessation of peristalsis is due to the complete stoppage of the arterial blood supply.

It is different when hemorrhagic infiltration of the intestinal wall has occurred. The blood current through the capillaries does not cease entirely, and even after thrombosis the tissue elements receive a certain amount of oxygen and nutritive material. To this is due the fact that the muscularis retains its contractility, and in the presence of accumulating carbon-dioxide it will therefore undergo severe convulsive contractions. Nevertheless, in some of these cases there occurs, sooner or later, a cessation of peristalsis, due to several factors. If the territory affected by thrombosis is very extensive there may be a severe and long continued lowering of the blood pressure, which will so damage the nutrition of the muscularis that its contractility is lost. The damaging effect of the lowered blood pressure upon the nutrition of the tissues is intensified by an edematous-hemorrhagic infiltration which occurs. An important factor is the excessive dilatation of the intestinal walls through the blood which may be extravasated in large amounts, and particularly on account of gas accumulation which may develop early. The intestinal microorganisms multiply rapidly after the extravasation of blood constituents and give rise to the development of gas in tbe intestinal contents of the horse, which are always subject to disintegration, and the gases are reabsorbed into the blood only insufficiently or not at all. Accordingly a circumscribed local meteorism forms promptly, and is promoted by irregular intestinal contractions which do not favor the propulsion of the contents.

The conditions for an active development of gases are, however, not present in mild forms of hemorrhagic infarction, hence no cessation of the erstwhile marked peristalsis occurs, if, after a certain interval, which is usually not long, the equilibrium of circulation is reestablished. On the other hand, grave forms of circulatory disturbances produce, in their further course, a gradual diminution of peristalsis which leads to complete cessation within several, occasionally within one to two, hours.

With the onset of circumscribed intestinal paralysis, peristalsis also ceases in the parts situated posteriorly, usually before the feces from the diseased portion have reached the rectum. The paralytic portion has the same effect as a portion obstructed from some cause or other, and as soon as its peristalsis has completely ceased, portions of the intestines anterior to it are stimulated to convulsive contractions by their accumulating contents; these are usually not very intense and not

very prolonged. Finally the increasing contents produce gradual cessation of contractility, which progresses towards the stomach.

The intestinal wall which is damaged in its nutrition, or eventually necrotic through grave circulatory disturbances, early permits intestinal bacteria to enter into the peritoneal cavity; these then multiply rapidly in a serous exudate and produce circumscribed, later general peritonitis, which causes continuous inflammatory pain; the bacteria and their toxins are also absorbed into the blood, general infection or intoxication with their symptoms follow; some of the symptoms seen in certain cases may also be due to bloating of larger portions of intestines.

What has been said in the preceding pages explains why thrombosis of mesenteric arteries, though common in consequence of the factors which cause it (sclerostoma), comparatively rarely lead to recognizable functional disturbances. The statement of Bollinger that three-fourths of all cases of colic are due to aneurysma of the mesenteric arteries is beyond doubt exaggerated.

Anatomical Changes. Aside from complications like enteritis or intestinal displacements those cases terminate fatally in which the circulatory disturbances are not compensated. In a certain portion of the intestines, generally in the colon, more rarely in the cecum, or in both simultaneously, or in the small intestines and exceptionally in the small colon, we find on postmortem examination the mucosa and frequently also the serosa dark blackish red, the mucosa forms pendulous projections, the submucous tissue and the neighboring mesentery are more or less infiltrated with a reddish-yellow serum and considerably thickened (1-3 cm). Similar infiltrations are seen on the exterior layers of the bowel-wall. The affected portion is, as a rule, sharply defined, both from the oral and from the caudal healthy portions, and the boundaries usually correspond to the territories supplied by arterial branches situated above the thrombosed point (see page 258). The intestinal contents in the affected portion and in different cases at a variable distance towards the anus appear dark tarry, bloody, sometimes only reddish and occasionally of normal color, but always thinner. The surface of the mucosa is covered by branlike dead shreds of epithelia, sometimes also with masses of fibrin. In grave cases the necrosis may have spread into the serosa and then yellowish spots can be seen from the outside. Exceptionally only do some portions of the intestine show a yellowish or greenish discoloration in consequence of anemic necrosis, as Casper observed in one case in a portion of the rectum, Puel in a portion of the small intestines and Marek in a portion of the pelvic flexure 15 cm. long. Cocu says that a hemorrhagic infarct of the intestinal wall due to thrombosis of the mesenteric arteries

can be easily distinguished anatomically from stagnation of the blood following dislocation of the bowel, because in the former the infarction is almost entirely limited to the mucosa, the muscularis being altered to a much lesser degree and the serosa showing only a few hemorrhagic spots at the most, as contrasted with the simultaneous infarction of the muscularis in cases of dislocation of the bowels. However, Cocu's assertion is not true in all cases. The distribution of the changes in the different layers of the intestinal wall depends solely upon the degree of the disturbance of circulation, which in either case may be equally high or low, although the disturbance in cases of dislocation much more frequently reach higher degrees. Yellowish or reddish-yellow serous fluid, often also a fibrinous exudate is found in the peritoneal cavity. The evidences of rupture of the intestines or stomach may also be present.

Then there are found thrombi or emboli in the trunk of the mesenteric artery or rather in the art. ileo-cœcolica, more rarely in one of the arteries of the colon or in the arteriæ jejunales, rarely in the posterior mesenteric artery; emboli are usually found in the smaller branches, especially when, according to Cocu, the mesentery or the wall of the large intestine is viewed by transmitted light or palpated. They will then be found as knots or elongated streaks the size of a millet seed or a pea. The blood coagula are sometimes fresh in appearance, rather moist and elastic, at other times drier and firmer, strongly adherent to the intima; sometimes a number of thrombi are found in the same vessels following each other. It occurs occasionally that a thrombus is not found on postmortem examination as it existed during life, because a movable occluding portion of the thrombus may have been moved by section of the affected vessel from the opening which it closed during life. The obstructed vessel, particularly often the anterior mesenteric or the arteria ileo-cœcocolica shows chronic arteritis with thrombosis and usually also aneurysmatic dilation. The blood coagulum usually contains the sclerostoma larvæ, sometimes 100 to 300 individuals, especially in younger animals, while thrombi in older animals contain few or even no larvæ at all. Sclerostoma larvæ are usually less numerous in city horses than in horses recently pastured (Wall).

Contrary to the view of Sticker that one finds larvæ in different stages of development according to the season in which the postmortem examination is made, Glage has shown that larvæ and mature worms may be found at all seasons in the intestines or in the mesenteric arteries of the horse. Stier also found larvæ in the thrombotic masses. They were much more numerous, however, in winter than in summer.

Symptoms. The disease sets in suddenly with symptoms of colic without any discoverable external circumstances; the animals may be at a meal, at work or at rest. The symptoms

of colic are either intense from the start, manifesting themselves in reckless rolling and throwing, or they are at first mild and reach a higher intensity later on, or the great restlessness first shown later yields to a more quiet behavior. The attacks of pain occur after shorter or longer, sometimes after very long, intervals.

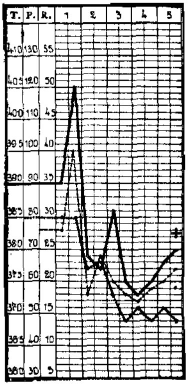


Fig. 18. Fever curve in thrombosis of the mesenteric arteries.

In the **mild type**, aside from the attacks of restlessness occurring at unequal intervals, a variable intensification of the intestinal sounds is observed in certain regions, sometimes over a large portion of the abdomen, and frequent defecation. The feces are normal except for their generally softened consistency.

Rectal exploration may reveal a pulsating dilatation or a trembling of the wall of the anterior mesentery artery or of one of its branches, occasionally also of the posterior mesenteric artery; the condition of the intestines and of the other abdominal organs is normal.

Pulse and respiration do not show any deviation in frequency and quality, the sensorium of the patients is perfectly normal in the intervals between the attacks of pain.

The restlessness becomes diminished, usually after a short time, always during the course of the first day and recovery occurs. In the **severe type** signs of restlessness are accompanied by unnatural positions, particularly in consequence of early bloating. The abdomen often becomes distended at the beginning of the affection. The intestinal sounds are loud in the beginning for one or two or sometimes for several hours; they become less frequent and disappear entirely. The percussion sound either remains normal or becomes louder.

After one or even several hours, constipation becomes complete and feces and gases are no longer discharged. The feces are softer and in very rare cases mixed with blood. The authors saw blood in the feces in only three out of more than 200 cases of thrombotic-embolic obstruction.

Rectal exploration shows a high degree of bloating in some portions of the intestines (meteorismus localis s. circumscriptus), usually affecting the colon, the cecum or both simultaneously or some loops of small intestines. The affected bowel is much dilated, its wall very tense, elastic and not painful. In bloating of the colon its left lower portion is enormously dis-

tended, not rarely reaches as high as the left kidney and displaces the left upper portion towards the median line or on the contrary towards the left, sometimes even more or less downward, especially when the cecum is bloated simultaneously. The pelvic flexure is displaced into the pelvis or into the right half of the abdominal cavity and the longitudinal bands of the left lower portion of the colon rnn from left to right, occasionally in a perfect spiral arrangement. The base of the bloated cecum reaches into the left half of the abdominal cavity and posteriorly into the entrance of the pelvis; the base of the cecum can be recognized as such by a longitudinal band running from the right flank at first backward, then downward and to the left, finally towards the thorax. The bloated loops of small intestines present as arm-thick tense, elastic, sausage-like formations, also the small colon; the latter, however, displays a longitudinal band along its surface. The other loops of intestines usually preserve their normal size until the end in contradistinction to the bloated bowels. The findings in the mesenteric vessels are the same as those in the milder form of the affection.

The pulse becomes weaker one to two hours after the onset *and rises to sixty beats per minute. Respiration and pulse become* accelerated and forced (in bloating of the large intestine the respiration is interfered with by the displaced diaphragm. The temperature (Figs. 18-19) is normal at the onset; it rises soon, however, in consequence of the appearance of complications [peritonitis, general sepsis, enteritis]). With a gradually increasing frequency and weakness of the pulse, dyspnea and stupor become more marked, muscular tremors make their appearance and are soon followed by death.

In rare cases the severe type of the disease takes a protracted course. If the circulatory disturbances persist for several days without reaching a very high degree, the absorption of intestinal gases will not be very much interfered with, and there is neither an abundant exudate, nor particularly active fermentation of the intestinal contents. Since the nutritive disturbances of the intestinal mucosa do not attain a very high degree, a diffuse peritonitis does not develop, and complete paralysis of the bowel either appears very late or not at all.

In cases of this kind restlessness may be observed, but rarely unnatural positions. The circumference of the abdomen is increased not at all or only very moderately. The intestinal sounds, which are at the onset very marked and of long duration, later on become less frequent without, however, ceasing entirely, therefore the expulsion of feces and gases is not entirely suppressed. The feces are sometimes hemorrhagic and fetid, and now and then intestinal gases are expelled sparingly. Rectal examination shows insignificant or moderate bloating, and in the further course occasionally the accumulation of more abundant masses of firm feces. Pulse, respiration and temperature show some deviation from the normal without, however, reaching

anything like what is observed in the severe type and in cases with a rapid course (Fig. 19). It also may happen that the temperature rises considerably at the beginning, and that pulse and respiration become quite markedly accelerated, to decrease considerably later on (Figs. 18 and 19). The sensory functions

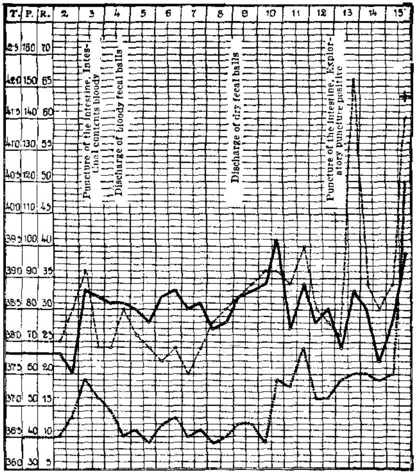


Fig. 19. Fever curve in obstruction of the mesenteric arteries. Fatal outcome. (Protracted course.)

are clouded, the appetite is suppressed or poor.

The clinical picture lasts sometimes for several days, exceptionally over a week. Friedberger observed in one case a duration of twenty-two days; Kremp one of twenty-one days; the authors saw in one of their cases a duration of sixteen days before the fatal termination. The protracted form may end in recovery or lead to death in consequence of enteritis, peritonitis or general sepsis.

In some cases displacement of the intestines is seen in either one of the different types. In severe cases acute dilatation of the stomach or rupture of the intestines may occur.

Course. The mild form of the disease lasts from a few quarters of an hour to several hours, rarely longer, never more than one day. The severe form has a duration of one to two

days, not infrequently only several hours; rarely more than two days and exceptionally two to three weeks, in the latter case it is characterized by attacks of colic which come on after long intervals. Sometimes mild cases of short duration follow each other at such short intervals (socalled habitual, periodic, recurring colic) that one may at first sight gain the impression that he is dealing with a single protracted attack.

The mild form ends in recovery, provided that a displacement of intestines or enteritis have not occurred; but such complications are rare in the mild type; the severe type usually terminates fatally. However, even cases which lead to hemorrhagic infarction may terminate in recovery, as has been shown by clinical observations in which a correct and reliable diagnosis has been made.

If an attack of colic has occurred, there is always the danger of recurrence with a final fatal termination.

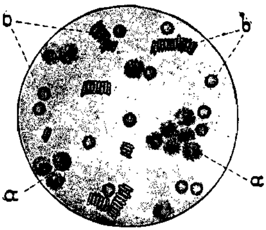


Fig. 20. Sediment of an intra-abdominal serous exudate in thrombosis of the mesenteric arteries—a, white; b, red blood corpuscles.

If an attack of colic has occurred, there is always the danger of recurrence with a final fatal termination.

Diagnosis. The history of preceding similar attacks without any apparent cause, excites suspicion of the affection. For the mild form of the disease, the occasional attacks of unrest are suggestive, also the fact that the peristaltic movements are unusually brisk, this being confined to certain portions of the abdomen, and further the absence of constipation and of general symptoms. In the grave type the erstwhile lively intestinal sounds becomes less frequent; after some time, constipation usually develops; rectal examination reveals localized meteorism. Within one to three hours after onset of the affection, gradually increasing symptoms of general affection develop. If an exploratory puncture of the abdominal cavity is made a yellowish or reddish fluid is usually obtained containing small particles of feces (Fig. 20), the punctured large intestine sometimes discharges a bloody, dirty red content, the feces voided in a natural manner may exceptionally be hemorrhagic. The more protracted cases are characterized by general symptoms of moderate severity, and careful general and rectal examination shows no other intestinal affection, such as stenosis, impaction or obturation. The presence of an aneurysm in a mesenteric artery cannot be used as a pathognomonic sign upon which to make a

diagnosis, since horses with such aneurysms may suffer from other diseases associated with colic. Nor has the finding of ova of sclerostoma in the feces of horses which suffer from colic any diagnostic significance since such ova are found in the feces of perfectly healthy animals.

Milder cases may be confounded with acute intestinal catarrh or with socalled convulsive colic, and the differentiation may not always be absolutely positive. The occurrence of symptoms of restlessness after errors of diet, after exposure to cold, lively intestinal sounds being audible over the whole of the abdomen, fetid soft feces with poorly digested particles of food point to a catarrhal origin of the colicky pains. If such symptoms are not present, however, the differential diagnosis may present great difficulty. The grave forms of mesenteric thrombosis may be mistaken for torsion, volvulus, incarceration, or primary intestinal meteorism. The two former conditions may be excluded by rectal examination, aside from a difference in anamnesis of the case, by frequently occurring, often continuous attacks of restlessness and by complete constipation, which usually supervenes very early. Primary bloating develops after the ingestion of easily fermenting food and is characterized by distension of all of the intestines. (Meteorismus universalis.)

Treatment. The establishment of a collateral circulation should be attempted by walking the animals and by the subcutaneous injection of camphor oil (every two to three hours 20 to 50 gm. up to 250 gm. pro die). Caffeine (5 to 10 gm.). Occasionally the intravenous infusion of physiologic salt solution is indicated. In view of the possibility of detaching portions of thrombi by increasing the velocity and pressure of circulation, and in view of the fact that a collateral circulation is easily established in the milder cases without any assistance from without, this procedure ought to be reserved for the grave cases, in which an additional embolism is a lesser evil than a continuous grave circulatory disturbance. Venesection with bloodletting which is in great favor with French veterinarians can only do harm, since it leads to a decrease of blood pressure.

Subcutaneous injections of morphine (0.4-0.5 gm.) or rectal injections of chloral hydrate (25-50 gm.) appear indicated in all forms of the disease, first in order to relieve abdominal pain, and second in order to suppress violent peristalsis, to prevent the possible occurrence of intestinal displacement or of rupture of the bowel. Breton recommends in colicky pains intraperitoneal injection of chloral hydrate 25 to 30 gm. in ten parts of sterile physiologic salt solution, but this procedure can be dispensed with and indeed it appears quite objectionable.

In the mild form one should abstain from the administration of any other drugs except narcotics. In the grave form puncture of the bowels should be made as early as possible.

Laxatives are useless and may be positively harmful. Mild

laxatives may possibly be given during the period of recovery.

Resection of a portion of intestine and suturing of the severed healthy ends may be tried in disease of shorter portions of the small intestine.

Prophylaxis. In order to prevent the ingestion of the larvæ of sclerostoma horses should be prevented from drinking dirty water from pools or marshes, and they should be kept away from wet and marshy pastures. During stable-feeding care must be taken so that the drinking water is pure and free from contamination with offal and feces. Sometimes water has to be made safe by filtering or boiling. Mickley has succeeded in the Beberbeck stud, where formerly many foals succumbed to the disease, in eradicating it entirely since 1899 by the interposition of filtering boxes into the system of pipes of the water works. Bang was able to stop the disease in the colts on a farm by having them watered at a well instead of at the water holes on the meadow where they had previously obtained their water in common with their mothers. In studs or in large stables where many horses are kept and where sclerostoma infection is frequent, all animals should be examined according to the suggestion of Albrecht for the presence of sclerostoma and those found affected should be separated and kept away from the common pastures or exercise ground until free from parasites. Removal of the manure and frequent changes of the bedding straw are also indicated since the floor and the walls of the stables where horses are kept frequently come in contact with their feces and since they therefore become contaminated with sclerostomata and their larvæ, they ought to be carefully cleaned and disinfected from time to time. Marshy rough feed from marshy pastures should not be fed to horses. French veterinarians consider alfalfa and clover hay as dangerous in this respect.

Whether the intravenous injection of atoxyl (100 gm. of a 3% solution), recommended by Dorn, is indeed valuable in eradicating sclerostomiasis in horses, has not yet been conclusively demonstrated.

Thrombosis of the arteria coeliaca. Thrombosis of the arteria cœliaca (see page 257), which is extremely rare, like thrombosis of the anterior mesenteric artery, does not usually produce any disturbances in general health. A case described by Friedberger (Z. f. pr. Vet.-Wiss. 1875, 258) shows, however, that such a thrombosis may also cause symptoms of disease. The celiac artery in the case referred to contained two half cylindrical thrombi 17 and 25 mm. wide, the central end of the thicker thrombus was free in the trunk of the art. cœliaca. After motion the horse presented symptoms of severe colic and from time to time made peculiar noises such as are heard when horses are vomiting; it was also attacked at times by tonic convulsions. These attacks could be produced at will whenever the animal was made to move. Eleven days after the last attack the animal was killed and

the postmortem examination showed a brown red discoloration of the gastric mucosa at the pyloric portion and signs of older hemorrhages in it, strong injection of blood vessels of the submucosa and hyperemia and hemorrhages in the first portion of the duodenum.

Thrombosis of Mesenteric Veins. Thrombosis of branches of the portal vein or of this vein itself has repeatedly been seen in domestic animals. Thrombosis of the portal vein is not uncommon in cattle and is caused by pressure of tuberculous masses or it may be due to injury by foreign bodies. Göhre and Spann have likewise observed thrombosis of the portal vein in cattle. Goubeaux, Collin, Mollereau, Cabaret, Kitt, Gratia have seen thrombosis of the portal vein of the horse; Conuochie, a thrombosis of the anterior mesenteric vein; Siedamgrotzky, a thrombosis of both veins of the colon; in a case of Barrier there was compression of the portal vein by a carcinoma of the stomach. The authors saw a case in a dog where the large veins of the mesentery were compressed by a tumor of the mesentery. Thrombosis of the mesenteric veins is not rare in deeper inflammations or ulcerations in the intestines (Kitt) and it is possible that coagulation of the blood in the veins may occasionally be caused by thrombosis of the mesenteric arteries.

Symptoms of disease are produced only by thrombosis or compression of the larger venous trunks, while thrombosis of smaller individual branches, or an incomplete closure of larger veins, remains unrecognized. The mesenteric veins show anatomical conditions similar to those of the mesenteric arteries, but they are much more easily dilated, hence the conditions for establishing a collateral circulation in thrombosis of the portal vein are quite favorable. Siedamgrotzky, Mollereau and Connochie saw attacks of colic in the horse, Göhre in cattle; the attacks extended over a period of three weeks in Siedamgrotzky's case. Cattle show under these circumstances signs of a grave indigestion in addition or to the exclusion of other symptoms (Göhre, Spann). The authors have seen diarrhea and hemorrhages from the intestines in a dog due to compression of several veins of the small intestines.

Postmortem examination either shows abdominal ascites or a hemorrhagic infiltration of the intestinal wall. Compression of the trunk of the portal vein not infrequently causes chronic interstitial hepatitis.

In the case of Huguenin, wedge-shaped atrophic infarcts of the liver with the apex directed against the hilus were caused by numerous emboli in the intrahepatic branches of the portal vein.

24. Internal Strangulation of the Intestine. Incarceratio et strangulatio intestini.

(*Darmeinklemmung, Darmverschnürung, Darmeinschnürung* [German].)

Internal strangulation of the intestines consists in a rapid closure of the lumen of the intestine by pressure from some body situated outside of the bowels, so that the strangulated or incarcerated portion of the intestine at once shows signs of stagnation of the blood.

Occurrence. Horses are more frequently affected by internal strangulation than other domestic animals. Of the cases

of colic in horses about 1 to 1.5 per cent are due to internal intestinal strangulation and of the fatal cases of colic about 5 to 13 per cent. Aside from the cases in oxen referred to above this form of displacement of the intestines is rare in cattle and other domestic animals, when one leaves out of consideration so-called internal hernia in cattle which occurs very frequently in oxen in some regions (according to Pedersen varying with the season, most frequently in spring).

Statistics of the Prussian army for the years 1892-1908 show that of 71,532 cases of colic among horses 1,069 cases (1.48%) were due to internal strangulation, and among these postmortem examination showed 304 (0.42%) to be diaphragmatic hernia; 234 (33%) displacements through the foramen of Winslow; and during 1892-1906 there occurred 218 cases (0.35%) of strangulation through clefts in the mesentery; 263 cases (0.43%) of strangulation by ligaments, pediculated tumors, etc. Among 8,426 cases of death from colic, 12.7% were due to internal strangulation.

In the Berlin Clinic there were, among 8,686 cases of colic which occurred from 1897-1907 ninety-three cases (1.1%) of internal strangulation, and among these thirty-four cases (0.39%) of strangulation in clefts or internal hernia; thirty-nine cases (0.45%) of strangulation by ligaments, etc.; seventeen cases (0.2%) of displacement through the foramen of Winslow. Among 1,408 horses dead from colic, 6.6% had suffered from internal strangulation.

The statistics of 1889-1894 and 1896-1908 of the Dresden Clinic show among 3,336 cases of colic, thirty-two cases (0.9%) of internal strangulation; among those four (0.12%) of diaphragmatic hernia; twenty-three (0.69%) of strangulation by ligaments, clefts, etc.; five (0.15%) of displacement through the foramen of Winslow. Among 500 horses dead from colic, 6.4% died of internal strangulation.

In the Budapest Clinic there were seen from 1900 to 1909 among 5,487 cases of colic, fifty (0.9%) due to internal strangulation. Among 725 horses dead from colic, 6.9% were due to internal strangulation.

At the Budapest Clinic internal strangulation of the intestines was noted in 75 out of 14,076 colic cases (0.53%) in the years 1900-19. During the same period this affliction was observed in 4.2% of 1,555 fatal cases of colic. At the Vienna Clinic, Wirth noted 3.3% in 10 years (1.1% displacement into the foramen of Winslow, 0.9% strangulation in the inguinal canal, 0.01% of so-called hernia of the diaphragm).

From the above data it appears that internal strangulation is comparatively frequent among cavalry horses and that among these cases those due to hernia of the diaphragm, to displacement into the foramen of Winslow and into clefts formed previously to the strangulation, are more common than they are among other horses.

Etiology. The intestines may become strangulated by various strands and bands present in the abdominal cavity. In this respect are to be mentioned congenital diverticula of the ileum (Meckels diverticula); exceptionally also acquired diverticula of the intestines (Kitt); the ligament between kidney and spleen (Marek, Forssell), the spleno-gastric ligament, the falciform ligament of the liver (Blanc), the urachus, the small omentum (Dupuy); pediculated neoplasms (generally lipomata) arising from the abdominal wall or the mesentery, possibly also from other abdominal organs, or exceptionally pediculated lobes of the liver (Pécus); in horses also exceptionally a stump of the spermatic cord, which has slipped back into the abdominal cavity, or the spermatic cord of an undescended testicle (Gutbrod); the larger omentum adherent to the anterior abdominal wall and twisted into a cord, sometimes the anterior mesentery itself (in a case of Dupuy and one of Prince, the cecum became

strangulated); finally postperitonitic connective tissue bands with one free end or with ends connecting various abdominal organs. Cysticercus tenuicollis caused strangulation of the duodenum in a hog (Späthe).

In other cases internal strangulation is brought about by portions of the intestines getting into congenital or acquired openings of the mesentery or of the omentum (Wiedemann observed a fresh strangulated rupture in the mesentery of a mare following parturition), or into torn and partially detached portions of the peritoneum or into a cleft between separated muscles (Roy). Some of the above mentioned ligaments may also tear and in this manner produce intestinal strangulation.

In a similar manner there develops in oxen or young castrated cattle (according to Mayr at the age of 3 months to 3½ years) what is called internal hernia (Strangulatio ducto-spermatica) a condition studied by a number of veterinarians, recently especially by Walch. This form of strangulation occurs only after castration with twisting or rather tearing off of the testicle, especially if the animals are castrated during the first weeks of life. Walch has also shown experimentally, that in such cases the reduplication of the peritoneum (pouch of Douglas) attached to the spermatic cord between the internal opening and the seminal vesicles, usually tears, so that loops of intestines slip into the opening so made, either immediately or later on. In other cases the stump of the spermatic cord retracts after tearing off its peritoneal covering, and gets from the spermatic canal into the abdominal cavity, or the spermatic cord is torn in its abdominal portion. In either case the remnants of the cord will either float as a free stump in the abdominal cavity or it will become adherent to the peritoneum and form a cleft. Since castration is performed according to different methods in various places, the affection only occurs in such regions where castration is performed as above described. Walch found this abnormality of the spermatic cord without strangulation in oxen in 60 to 70% of the slaughtered animals. Tearing of the fibrous tunic of the external inguinal ring by a strong pull can also be detrimental if the spermatic cord is severed transversely, and if the testicle and vessels are then pulled off. A case of strangulatio ducto-spermatica in a horse has been described by Janson; in this case the pouch of Douglas had likewise been torn. Metzger and Strauss have exceptionally seen the affection in sheep. Tearing of the reduplication of the peritoneum in sheep and in other animals is rare, since this fold of peritoneum is more resistant and smaller than in cattle.

Socalled internal-hernia will lead to intestinal strangulation, either from the start or later on. Diaphragmatic hernia occurs particularly in the horse, more rarely in cattle and dogs. It is either congenital or formed during extrauterine life, the diaphragm rupturing in consequence of too great, sudden intra-

abdominal pressure, or the accident may occasionally be due to dilatation of the stomach or to bloating. Such ruptures are most common in horses which have to jump (hence they are most frequently found in army horses). Displacement of the intestines into the foramen of Winslow occurs exclusively in the horse and the intestine then gets between the layers of the great omentum (enterocele omentalis). Jöhnk observed a case of this form of displacement of the intestine, but what Lucet & Bru have described as an enterocele omentalis in cattle, was, according to Mathis, only a strangulation into a tear of the omentum.

Incarceration of external hernia, which is a surgical lesion, leads to the same local symptoms and to the same clinical picture as internal strangulation.

Among the enumerated causative factors are some (incarceration in narrow clefts) which are able to cause strangulation at once, but additional predisposing causes are, as a rule, necessary to bring this condition about. A slight filling of intestines enables loops to slip into a preformed opening: in this condition the intestines can also easily change their position, and openings usually covered by the large intestine (foramen of Winslow, tears in the lower portion of the diaphragm) can now become accessible to the small intestines. Active peristalsis, whether it be due to external or internal cooling, catarrhal affections, embolism, etc., strong action of the abdominal press (pulling of a great weight, galloping, jumping, walking uphill, efforts, rolling, falls, being run over), favor the displacement of loops of intestines into a cleft during energetic movements of the body, or pediculated formations or bands which are free at one end may in rolling become twisted around loops of intestines. Under similar conditions strangulation is produced by the spleno-nephritic ligament, a variety of strangulation which is not rare. (In five cases observed by the authors the left portion of the colon was always strangulated.)

The jejunum, which is most motile and most slender, is strangulated most frequently; other portions of the intestines are less commonly affected in this manner.

Pathogenesis. If the opening into which a portion of intestines has become displaced is narrow compared with the caliber of this part of the bowel, or if a band-like body has been twisted around the intestines, the intestinal wall and its mesentery are compressed to a certain degree and the lumen becomes closed. In other cases the pressure becomes effective only if the other additional factors mentioned above have caused further portions of intestines and mesentery to become displaced, or after the portion of the intestine that was first displaced has become bloated in consequence of kinking or has become swollen in consequence of venous (passive) congestion.

The outflow of venous blood from the affected intestine and

its mesentery is interfered with in proportion to the pressure exerted, while the thick-walled arteries with their high internal pressure keep on pumping blood into the tissues, possibly in a somewhat decreased amount. Hence, stagnation of blood (stasis) soon occurs in the incarcerated portion of intestines and also, owing to an increase in venous pressure, a diminution of the velocity of the entire blood current with all the consequences which have been discussed in detail under the head of thrombosis of the mesenteric arteries (see page 258). At the place of strangulation the compression produces an anemic ring.

The rapidly increasing stasis, and the overloading with carbon-dioxide due to it, excite strong, frequent and occasionally convulsive contractions in the strangulated portion of intestines, causing colicky pains. The frequency and the intensity of these convulsive contractions may be the greater, the longer is the portion of intestine which is strangulated. A stronger pressure, which, however, does not completely shut off the arterial blood supply, clearly causes a considerable overloading of the blood of the affected portion with carbon-dioxide, and causes stronger contractions. Everything else being equal, strangulation of the small intestines which are rich in nerves causes more intense abdominal pain than strangulation of other portions.

Strong contractions may cause the entrance of more loops through the place of strangulation by suction or traction if the part situated towards the anus becomes distended, so that it pulls on the part situated more towards the stomach (Wilms). Throwing down or rolling with suddenly increasing abdominal pressure may, on the other hand, force more loops of intestines through the place of strangulation by pressure from behind (Kertész).

The strangulated portion of bowel, which is filled with intestinal contents mixed, with extravasated blood, will soon become bloated, because the gases can neither be removed towards the stomach nor towards the anus, nor can they be absorbed by the blood. Peristalsis and pain finally cease in the strangulated portion after it has been dilated ad maximum and after edematous infiltrat[illegible]ed. Peristalsis of the portions situated between the strangulation and th[illegible] almost at once or shortly after the strangulation occurred, partly by reason of the cessation of the movement of intestinal contents through the strangulated portion of the intestine, but especially through inhibitory reflex action by way of the sympathetic nerves, originating from the bruised nerves at the point of strangulation, which can be shown in animal experiment (Lehmann, Mentzer) and which simultaneously also bring about an inhibition of the movements of the rectum with convulsive contractions of the internal sphincters. Contractions persist for a long time in the portions situated anteriorly to the point of strangulation, and some may later become convulsive and cause pain; but even here, sooner or later, they will gradually close, partly by reason of reflex inhibition, but especially because of the gradual distention of the intestines with stagnated liquid and gaseous contents.

The gradually increasing swelling of the intestinal wall and of the mesentery, and the involvement of more loops of intestines produce increased pressure upon the sensory nerves that are caught in the place of strangulation; this causes a continuous, but not severe pain, as experiments on horses show; the latter may even be entirely absent where the strangulation is not too severe. In the further course continuous pain, which increases on pressure, is also caused by peritonitis which develops. The general symptoms are similar in origin and character to those encountered in thrombosis of the mesenteric arteries (see page

265); however, they vary in different species of animals as to their appearance, according to variability in susceptibility to microorganisms and their toxins. The shorter the strangulated portion of intestine and the less firm the strangulation, the later and the less severely will these general symptoms appear.

Anatomical Changes. The strangulated portion of intestine appears, as a rule, dark to blackish red, distended and tense, the wall thickened by serous-hemorrhagic infiltration, friable, the intestinal lumen filled with a thin fluid, fetid, hemorrhagic mass, the mucosa blackish red, on the surface dirty yel-

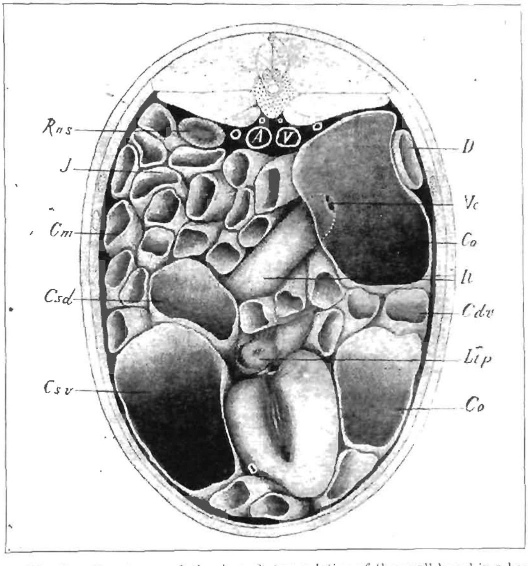


Fig. 21. Fecal accumulation in and strangulation of the small bowel in a horse. Oblique section of the abdominal cavity, above through the 3d lumbar vertebra; below through the ileum. *Rns* left kidney; *J* loop of jejunum; *Cm* loops of the small colon; *Csd* left upper, and *Csv* left lower colon; *D* duodenum; *Co* cecum; *Vc* opening of the ileum into the cecum; *Il* ileum; *Cdv* right lower colon; *Lip* pediculated lipoma, the pediculum of which has strangulated a loop of the jejunum.

lowish, smeary. The site of the strangulation is indicated by a pale yellowish strip; sometimes the strangulated portion shows several segments, separated from each other by pale rings with

alternating reddened portions (gradual entrance of the strangulated portion in sections). Aside from strangulation a circumscribed or diffuse peritonitis, and perhaps intestinal rupture may be found. The peritoneal cavity contains a yellowish or reddish serous exudate, frequently mixed with shreds of fibrin. Lesions of sepsis or sapremic intoxication may also be present.

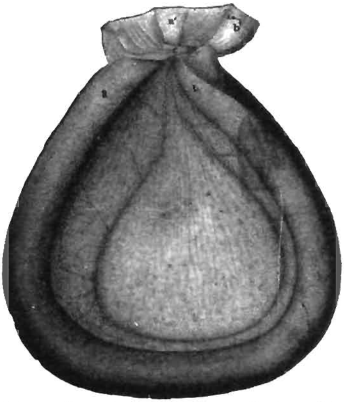


Fig. 22. Loop of small intestine (*a*, *b*), strangulated in a cleft of the mesentery (*c*).

Symptoms. In horses, sudden, rarely gradually developing symptoms of violent abdominal pain are observed; in some cases the animals previously show the symptoms of stenosis of the intestine. Usually the animals throw themselves down recklessly, roll, kick with their feet, and their restlessness may even assume the character of a maniacal excitement. Later they avoid reckless throwing; they lie down, but do so carefully, or they stand still, probably in consequence of the development of peritonitis, which causes pain. Rarely do the animals behave quietly and with care from the start, and do not throw themselves down, and in such cases one might think of a stronger constriction of intestinal and mesenteric nerves at the place of strangulation. Sometimes a reckless behavior persists until the end. Stretching, lying down on one side, squatting dog fashion on the haunches, kneeling, lying on the back are observed frequently; these positions have, however, no particular significance. The restlessness may be almost absolutely continuous, so that the patients are still hardly a moment, either in standing or in the recumbent posture, or the restlessness comes on in short attacks; this depends upon the intervals at which the convulsive intestinal contractions occur. In strangulation of the rectum or small colon, strong pressing down is seen occasionally. The expression of the face betrays distressing pain the same as in intestinal displacements in general.

The abdominal circumference is rarely increased, and if at all only moderately so. Intestinal sounds may be increased for some time in strangulation of the rectum or small colon; in

other cases they soon become less frequent and are eventually suppressed.

Defecation ceases at once or soon; in exceptional cases, when the strangulation is not complete, there may be only retarded defecation in the further course of the disease.

Rectal examination shows that the rectum is empty and frequently the strangulation of some parts of the intestines, its seat and its origin, can be ascertained. In strangulation of the rectum the hand meets an impediment and it is frequently impossible to pass even one finger. The wall of the intestine is folded in front of the obstruction and is tender; if it is possible to press the finger through the impediment this is found coated with a dirty, reddish, fetid fluid. In strangulation of any other portion of the intestines one can ascertain that there is somewhere a much bloated intestinal portion which decreases in one place to a folded string; here the tissues are tender and surrounded by a tense ring. Sometimes one finds pediculated tumor-like formations, and occasionally it is even possible to determine the direction in which the constricting band or the pediculated body has become wound around the bowel. If the place of strangulation is in the anterior portion of the abdomen, which cannot be reached by the hand, it is possible at most to demonstrate circumscribed bloating and tenderness of certain portions of the intestines, or the examination may at first be perfectly negative while later on a gradually increasing bloating of moderate degree will be noticed of the parts situated in front of the strangulation towards the stomach.

In strangulation of the intestines by the nephro-splenic ligament, one can ascertain that the spleen is not in contact with the left abdominal wall as usual, and its base has been displaced far downward, approximately to the middle of the left abdominal wall, while the ligament has been drawn out to a cord 15-20 cm. long. In the ring which is not closed entirely and is formed by the nephro-splenic ligament, the free upper end of the base of the spleen, which is usually horizontal, by the left kidney and the left abdominal wall, one finds the left portion of the colon suspended on the nephro-splenic ligament; the upper left loop of the colon usually rests directly upon the ligament and appears strangulated, while the lower left loop which is now directed upward and to the left is not compressed or very little. The posterior half of the bloated left portion of the colon runs backward and downward in a curve, and the upper loop is more or less situated in front of the lower loop. If at the same time the pelvic flexure is directed forward and somewhat laterally, the bands of the lower portions present a spiral course. Strangulation of loops of small intestines, which the authors have seen occasionally in other forms of colic, was never found in this affection.

According to Larsen the presence of some tense loops of small intestines in the region of the right flank, directly at the abdominal wall, points to displacement of the small intestine through the foramen of Winslow; this was also found in a case of Forssell. The authors have had similar findings in displacement of the small intestines, due to dilatation of the stomach.

In diaphragmatic hernia respiration is difficult from the start on walking downhill, and in certain positions on the side Over the posterior and lower portions of the thorax one finds tympanitic and often metallic sounds, which frequently change in pitch. Again a periodically changing or bilateral dullness may be found if the prolapsed abdominal organs have been partly

displaced into the thoracic cavity through a rupture of the diaphragm (Moeller). Soon there appear symptoms pointing to acute pleuritis (tenderness on pressure upon the intercostal spaces, friction sounds), or to accumulation of blood in the pleural cavity. In diaphragmatic hernia of the left side the heart may become displaced by prolapsed loops of intestines and the apex beat may disappear. Bigot observed a transient intense edema from the shoulder to the sternum. Floquet heard intestinal sounds transmitted by the trachea in a case of strangulated diaphragmatic hernia. In cases of diaphragmatic hernia rectal examination is negative.

A peculiar type of dyspnea was seen in a horse where a recent extensive rupture of the diaphragm gave rise to a prolapse of the small intestines and the stomach. In spite of marked and rapid excursions of the ribs a passive drawing in of the intercostal spaces did not occur, but, on the contrary, a strong inspiratory drawing in of the epigastrium, the flanks and the region of the loins. The cause of this form of dyspnea was found in the large extent of the rupture of the diaphragm; during each inspiration a part of the small intestine was drawn through the opening of the diaphragm into the thorax and the intraabdominal pressure was diminished in consequence.

In strangulation of the small intestine acute dilatation of the stomach (see page 137) is frequently seen later on.

The behavior of the pulse, sensorium, temperature and respiration are similar to what is seen in grave forms of embolism and thrombosis (see page 266); the pulse rises after a few quarters of an hour, or at least after a few hours, to above sixty per minute, and as it grows faster it grows weaker, the sensorium becomes clouded, respiration difficult and the temperature rises.

In strangulation of a very short piece of intestine, it may occur, however, that acceleration of the pulse and elevation of temperature takes place very slowly and does not reach a high degree. In a horse where a piece of small intestine 30 cm. long became strangulated by a pediculated lipoma, the pulse was only 56 after 13 hours and the temperature 38.6%, yet, when a laparotomy was made 2 hours later, fetid fluid was found in the free abdominal cavity and necrosis of the intestinal wall.

The clinical picture in **cattle** is also characterized by marked symptoms of colic. The animals kick with their hind legs and push against the abdomen with their horns; they shake their heads, look around towards the abdomen, lie down, soon get up again, trip restlessly from place to place and sometimes step into the crib. The gait is sometimes stiff, because the right hind foot is not placed sufficiently forward. The restlessness ceases after several, usually after 6 to 12 hours; it may, therefore, happen that, if the attack occurred in the evening, it is over the next morning, and that only the condition of the bedding straw shows that the animal has been restless during the night (Walch). Symptoms of restlessness are absent during the further course, and with the deterioration of the general condition one observes only groaning and slight restlessness at longer

intervals, due to the peritonitis which has developed. Perspiration is frequent at the onset of the disease.

The circumference of the abdomen is at first not changed; later on moderate bloating occurs, due to moderate dilatation of the portions of intestines nearer to the stomach, and also on account of secondary bloating in the rumen. The intestinal sounds are suppressed and cease entirely later on; sudden pressure on the right half of the abdomen later produces splashing sounds and pressure upon the abdominal wall, especially on the right flank, often produces symptoms of pain. After restlessness has set in the feces are voided for a short time, for a few hours at the utmost, and afterwards complete constipation supervenes; perhaps very little fecal matter or some mucus may be expelled with great efforts.

Rectal examination, which, according to Walch, is best made on the standing animal, will always (Walch) reveal the spermatic-duct type of strangulation (strangulatio ducto-spermatica), and freqnently also the other types. The rectum is found empty. In strangulation of the spermatic duct type, one finds a tumor, the size of a fist up to the size of a child's head, occasionally as big as an adult human head, in the neighborhood of the internal inguinal ring, as a rule on the right, very rarely on the left side. The tumor is composed of tense dilated intestinal loops and is adherent at one point to the abdominal wall, where one can feel a tense cord. If the retracted spermatic cord has become wound around the intestines, the tumor is more motile and is nearer to the median line or in the middle of the anterior margin of the pelvis; in this case one can also feel the strangulating cord. In strangulations of other types the findings in cattle are the same as they are in horses. In a case of incarceration of the small intestine, Jöhnk palpated with the hand introduced into the terminal loop of the colon, finding a painful tumor, rounded and as large as a fist, which was movable with the colon.

In spite of the fact that the animal has subsequently quieted down, the sensorium becomes clouded. There is complete lack of appetite and great weakness, so that the animals are no longer able to get up.

The pulse early becomes rapid and weak, and it rises to 120 to 130 per minute; simultaneously with this acceleration, the peripheral portions of the body become cool, the visible mucosa, which is at first reddened, becomes pale. The respiration is accelerated from the start and becomes more difficult. The elevation of temperature which sets in later, announces the development of peritonitis or of general infection.

Dogs become depressed, they lie around a good deal, sometimes they cry out, groan, whine, constantly change their place, sometimes look around towards the abdomen, get np suddenly, roll, stretch their extremities stiffly, or remain for a long time resting on their abdomen. They frequently exhibit an irritable

temper. Gagging or obstinate vomiting are seen frequently. Constipation is complete from the start.

The abdominal circumference is at first normal, it may become larger in the further course; the abdominal walls are tense, and strong pressure at certain points excites pain. In palpation of the abdomen, which must be made under a slight narcosis if there is great tension, one can feel somewhere in the abdominal cavity, distended painful loops of intestines; sometimes the strangulation can be mapped out as a firm, stretched painful mass.

The restlessness, which is not very great, decreases within the next hours, but the deterioration of the condition is shown by increasing depression, weakness, elevation of temperature, a weak and rapid pulse, continued obstinate vomiting and constipation. As in serious diseases of dogs in general, one may in this case observe a lowering instead of an elevation of temperature.

In a dog with a recent, not incarcerated diaphragmatic hernia, Steinberg noted severe dyspnea which diminished when the fore part of the body was placed high, but was increased very much when the posterior part was raised. The dyspnea disappeared when the thoracic wall had been fixed by cloths wound firmly around the chest and the fore part of the body had been raised; but it returned six weeks later when the dog happened to be held fast by the tail and made energetic efforts to free himself.

In **hogs** Späthe described a clinical picture in intestinal strangulation similar to that observed in dogs.

Course. The disease is characterized in all species of animals by a sudden onset; sometimes it follows upon a manifest predisposing cause, which is preceded in infrequent cases by the symptoms of intestinal stenosis. Pain is either intense from the very onset or it may increase gradually, but rapidly; this depends upon whether the strangulation has been produced suddenly or has become complete only after some time. Severe pain persists as long as the incarcerated intestines retain their power to contract; according to variability in the degree of strangulation this may last from a few hours to one day. Later on the animals become apparently perfectly quiet or only occasionally exhibit some signs of abdominal pains which are caused by contractions of the intestines situated between the place of strangulation and the stomach, or by peritonitis which has set in in the meantime.

If the animals are left to themselves they soon perish in consequence of rupture of the intestines, general sepsis or intoxication. Horses usually, dogs frequently, succumb during the first day while cattle remain alive for two to six days (Walch). In one case a horse with a strangulation of a piece of the ileum 30 cm. long through the great omentum died after 5 days.

The possibility in the beginning of the affection of a spontaneous reduction of the strangulated portion of intestines must be admitted in exceptional cases. In the case of Renele spontaneous rupture of the spermatic duct occurred in a bull that made strenuous efforts to free himself from the shackles which had been placed upon him preparatory to performing laparotomy.

Diagnosis. Internal strangulation of the intestine can be diagnosticated with certainty only if rectal examination or palpation of the abdomen furnish a positive result; all other symptoms can at best furnish a more or less well founded suspicion. If there is good ground for a strong suspicion an exploratory laparotomy should be made in cattle and in dogs, occasionally also in other animals; this enables us to determine, by the aid of the hand which is introduced through the laparotomy incision, the seat and origin of an existing strangulation. In steers less than 9 to 10 months old, where a rectal examination cannot be made, an exploratory laparotomy should be made in all cases when sudden symptoms of colic have come on with constipation, and where treatment during a period of twelve hours has not brought about any improvement (Hoffmann). If there are symptoms pointing to obstruction of the intestines and one hears over the posterior, lower parts of one side of the thorax tympanitic or possibly metallic percussion sounds which are variable in pitch and intensity, if quite intense intestinal sounds are audible in this region, and if symptoms of beginning pleuritis or hemothorax appear soon, then a diagnosis of incarcerated diaphragmatic hernia appears safe, especially after an exploratory puncture of the thorax has furnished hemorrhagic intestinal contents. Aside from strangulation, circumscribed intestinal meteorism is encountered also in torsion of the intestine, in the grave form of thrombosis, in acute dilatation of the stomach, hence this can only be utilized as a suggestive symptom in connection with other symptoms, and with certain circumstantial evidence in those cases where the exact site of the incarceration cannot be felt or otherwise ascertained directly. However, if dealing with a strong meteorism localized to a few loops of intestines, one is seldom in error in thinking of intestinal strangulation, torsion or volvulus. The clear, yellowish or slightly reddish serous fluid, which is at first obtained by exploratory puncture of the abdominal cavity, is found in internal strangulation, torsion, invagination, and in the graver forms of thrombosis of the mesenteric arteries.

Acute primary dilatation of the stomach of the horse can be excluded with certainty, in the majority of cases, by anamnestic data, such as early difficulty in respiration, absence of early and marked disturbances of defecation and rapid improvement, and definite recovery after the proper use of the stomach tube. Torsion, volvulus and invagination can only be differenti-

ated from internal strangulation by the findings on rectal examination. It is not of very great importance if it is impossible to differentiate the various forms of intestinal displacement, since the treatment is the same in all three forms. (In the case of Landenberger presumably intestinal strangulation through the persisting urachus, the rectal examination was similar to that observed in strangulation of the spermatic duct). Incarcerated external hernia may be recognized on the basis of the symptoms of closure of the intestines in connection with the local signs; hence an examination of the inguinal ring and the scrotum should always be made whenever there are colicky pains, particularly in stallions. Changes of position and internal strangulation may be confounded with those types of intestinal obturation (impaction) which cause almost continual pain and tenderness upon pressure in well defined places of the abdominal cavity (see page 243). Similar symptoms have occasionally been observed in cattle in obturation of the bowel by blood coagula. Aside from the result of rectal exploration, the continuous absence of general symptoms and anamnestic points frequently furnish sufficient diagnostic data. Obstruction of the urethra by calculi in male cattle is distinguished from intestinal strangulation by the following features: Urination ceases in obstruction of the urethra, but not the defecation which goes on undisturbed, the patients exhibit peculiar pulsating motions of the urethra in the region of the intestines, rectal exploration in the inguinal regions is negative and one can only feel the urinary bladder in the shape of a flat cylindrical, thick tumor, lying exactly in the median line. In exceptional cases torsion of a cryptorchitic testicle might cause difficulties in diagnosis, if it produces colicky unrest, as happened in the case of a horse; on rectal examination, the cord with its cryptorchitic testicle was felt above the inner inguinal ring; it was changed into a thick, firm, painful rope, with spiral form. Joehnk observed a case of torsion of the testicle in a hog and one in a young bull, and in both symptoms of unrest were absent.

Prognosis. The prognosis is unfavorable if rectal or operative correction of the intestinal strangulation can, for some reason or other, not be accomplished, because spontaneous recovery occurs only very exceptionally if at all, and medicinal treatment has no influence whatever upon the affection. The operative procedure is determined preferably by the species of the patient, also by the duration of the train of symptoms, the general condition of the animal, the condition of the constricting body, and by the question whether reposition can be accomplished per rectum or only by a laparotomy. The prognosis is most favorable in cattle, even in such cases where a laparotomy is necessary. (Hoffmann observed losses only in 5% out of 185 cases.)

Dogs likewise stand the operation of laparotomy well. The

presence of a secondary peritonitis or of grave disturbances of the general condition very much diminish the chances of operative procedure, and these are abolished completely if symptoms of collapse (unconsciousness, pale mucosæ, subnormal temperature, unrecognizable pulse, paralytic weakness) and high fever in horses are already present. Exploratory puncture may furnish certain data of prognostic value; if a putrid smelling fluid is obtained from the abdominal cavity there is no more hope of saving the animal.

Treatment. Recovery can be expected only after operative interference with reposition from the rectum or after laparotomy; medicinal treatment must be limited to the administration of narcotics (morphine, chloral hydrate) since laxatives are not only useless but may rather be injurious. In case of great restlessness in cattle chloral hydrate could also be injected intravenously (0.09 gm., at the most 0.12 per kg. body weight, in 5% solution; narcosis lasts about 60 minutes; no bad effects on the milk and meat). Blume recommends repeated subcutaneous injections of atropin in cases of displaced intestines (0.05 gm. for horses, 0.1 gm. for cattle).

In certain cases in horses and cattle, the reduction per rectum is possible liberating from the rectum loops of intestines that are strangulated by bands or pediculated tumor, either by untwisting a band or by tearing it or the slender pedicle of the tumor. Perl succeeded in tearing with his spread fingers the pedicle of a lipoma, the pedicle having strangulated the rectum (the authors attempted the same procedure in two cases of strangulation by pediculated lipomata, but they were not successful).

Strangulation of the intestines by the nephro-splenic ligament may be overcome in horses in the following manner: The right hand is introduced into the rectum of the standing animal, and it is then pushed between the stretched nephro-splenic ligament and the compressed bowel; the latter is then grasped to the right and below and is pushed towards the left, being at the same time pressed down with the thumb. While executing this rotating motion towards the left, the back of the hand attempts to displace the base of the spleen towards the median line of the abdominal cavity. The intestines can usually be released within fifteen or thirty minutes, occasionally within a few minutes. In four of the authors' five cases, they succeeded without trouble, but in one case a laparotomy became necessary Forssell has relieved cases of this type by this method; he placed the patients on the back. This position is also deemed necessary by Nordquist in case reposition of the strangulated colon in the standing animal by lifting it upward should fail.

In spermatic duct strangulation of oxen one may, if the condition has not lasted long, attempt to press the intestines which are not yet bloated through the strangulating ring. As a rule, however, the animals are only seen after they have been sick some time, then the spermatic cord must be separated or torn loose. After the posterior portion of the abdomen of the patient has been elevated, the well-greased arm is introduced into the rectum up to the elbow, then one attempts either to separate the duct or to tear it loose. The operative procedure is variously described by different authors. Walch grasps the cord between thumb and index finger, then closes the hand and turns it downward and to the right, and also draws it backwards and to the left.

Servatius grasps the cord from above, from right to left and winds it once around the index finger in a spiral; he then closes this finger and presses it against

the thumb. Then he pulls towards the abdomen and tears the cord away from its attachment. In animals more than two years old, when the attachment of the spermatic cord is much firmer, he attempts to grasp it with the whole hand, brings it to the back of the hand, winds it around the wrist and now tries to tear it loose with one strong pull; if possible an assistant pulls at the same time on the scrotum.

Hoffmann draws the cord with the tip of the fingers or with the whole fist from in front upwards and then from within backwards into the middle of the pelvis; the cord is usually torn during this manipulation. If, however, a tear does not occur, he winds the elastic cord from one to three times around his hand and changes the direction of the traction backwards, upwards and to the left.

The described procedures usually succeed in relieving internal strangulation in oxen. (Probst succeeded in twenty-seven cases out of twenty-nine, Pedersen in 60 cases out of 61.) In 1 case observed by Poeschel, the freed intestinal loop continued to remain in a relaxed condition, thus causing a paralytic ileus. The practice of driving the animal down hill, drawing it up by the hind legs, rolling it over the back, etc., which still prevails here and there, hardly promises success. The affliction can be prevented by the proper instruction of the owners on the castration of male calves.

In smaller animals or in calves less than 9 to 10 months old, laparotomy becomes necessary if the animals are to be saved. In the case of Humann the reposition of a displaced intestine was possible by the repeated and copious infusion of warm water into the rectum while the hind part of the calf was elevated. This operation likewise is usually necessary in horses, and also in cattle in all other forms of strangulation except spermatic-duct strangulation; in oxen older than one year, the latter rarely necessitates laparotomy. It is obvious that one must not wait with laparotomy until necrosis of the intestinal wall or a diffuse peritonitis has developed. The abdominal incision is to be made as near as possible to the location of the strangulation; the strangulating band must be severed, but clefts should be enlarged by a bloodless method, that is, by stretching or tearing. In this manner recovery has frequently been brought about in internal hernia of cattle and occasionally, also in other types of strangulation of this species (Kränzle, Schiel, Jöhnk).

In order to prevent relapses in cases of strangulation of the spermatic duct, which may occur when the spermatic cord is simply loosened or hanging free in the abdominal cavity, Dierick recommends laparotomy with resection of the spermatic cord at the point where it has torn away from the peritoneal fold.

Plósz & Marek and also Teltsch have performed laparotomy in the horse, each in one case of intestinal strangulation; the band was severed with the aid of a pair of scissors introduced into the abdominal cavity. Rogerson, however, did not succeed in relieving a strangulation caused by a tumor. Forssell made an interesting report of the details of three cases of laparotomy performed in horses for the relief of strangulation of the intestines through the foramen of Winslow. One of the cases terminated in recovery, two of the horses died, one of them from hemorrhage due to injury of the portal vein.

25. Volvulus and Torsion of the Intestine. Volvulus et torsio intestini.

Volvulus and torsion of the intestine are a rapid closure of the bowel lumen, caused by a turn of the intestine around its own axis or by strangulation caused by other loops; simul-

taneously with it stagnation of the blood occurs in the affected portion.

On the basis of this definition postmortem torsion, which is comparatively frequent, must be separated from volvulus and torsion; and also displacements of the intestine which are not really pathologic in character and occur quite commonly when certain portions of the intestine are distended with masses of gas or feces, which are, however, not accompanied by an encroachment upon the intestinal lumen, nor by a stagnation of blood in the intestinal wall.

Occurrence. Volvulus and torsion of the intestine occur with rare exception only in horses. About 2-5.5% of the colics of the stomach and intestines are due to this cause. Oppenheim, Reichert, v. Ow, Guittard and Joehnk have each seen one case in cattle; Ries two cases; Hess saw torsion of the cecum in a calf four days old. Glage saw a few cases of torsion of the cecum in hogs. Liénaux and Probst each saw a case of volvulus in a dog; Johne a case in an elephant.

Volvulus and torsion in horses are not seen everywhere equally often. This depends upon a difference of susceptibility in various breeds and on differences in the attendance and in the use of horses in different countries. Torsion of the cecum is most frequent in countries and states where the heaviest horses are kept (the affection is more frequent in South Germany than in North Germany). An increase of the number of cases of torsion of the cecum has been noticed more recently and is probably due to the fact that more attention has recently been directed towards torsion of the cecum, which is not always easy to detect even anatomically.

Of 98,249 cases of colic in the horses of the Prussian army in the years 1892-1912, 3,618 cases (3.68%) were due to volvulus and torsion. Of these there were found 1,697 times (1.72%) mostly volvulus and only seldom torsion of the small intestines, 21 times (0.02%) volvulus and torsion of the small colon, 1,787 times (1.82%) volvulus of the large colon, and 113 times (0.11%) torsion of the cecum. In 8,426 cases of death from colic this form of intestinal displacement was represented in 34.5%; among these volvulus and torsion of the small intestines in 16.1%, torsion of the colon in 18.2%.

In the Berlin Clinic there were 8,686 cases of colic (1897-1907), and 437 of these (5%) were due to volvulus or torsion; volvulus or torsion of the small intestines or of the small colon occurred 183 times (2.1%); torsion of the large colon 242 times (2.8%) (in four cases of the colonic flexure); and there was one case of torsion of the cecum. In 1,408 fatal cases, displacement of the small intestine and small colon was represented with 13%, and displacement of the colon with 17.1%.

During the years 1889-1894 and 1896-1908, 3,336 cases of colic were seen at the Dresden Clinic; among these were 183 cases (5.5%) of volvulus and torsion; sixty-five cases (1.95%) were volvulus of the small intestines; 117 cases (3.5%) torsion of the colon and one case (0.03%) torsion of the cecum; 36.6% of the deaths from colic were due to this form of intestinal displacement and among these 23.4% to torsion of the colon and 13% to volvulus of the small intestine.

At the Budapest Clinic 14,076 colic cases were observed in the years 1900-19, of which 510 cases (3.6%) were due to volvulus and torsion. Among the latter there were 210 cases (1.49%) of volvulus or torsion of the small intestine, 314 cases (2.23%) of torsion of the large colon, 3 cases (0.02%) of torsion of the cecum, and 3 cases (0.02%) of torsion of the small colon. Of the colic cases terminating fatally 18.8% were due to volvulus or torsion of the intestines. At the Vienna Clinic, Wirth found in 10 years as the cause of death in colic 21.4% of cases due to torsion of the large colon, 23.2% due to volvulus of the small intestine and 0.8% due to torsion of the cecum.

Etiology. Volvulus and torsion of the intestines are without doubt brought about in a majority of cases by mechanical

causes. Experimental proof of this statement has been furnished by observations that animals acquired this intestinal displacement immediately after certain mechanical injuries, and died from it (Mayer, Glage, Liénaux), or that intestinal torsion occurred secondarily, in consequence of rolling in the course of other painful intestinal affections or displacements (strangulation, invagination), (Rulf, Jöhnk, authors' observation). The importance of mechanical factors as a causative agent is also proven by postmortem displacements which are seen particularly often if cadavers of horses have been transported before postmortem rigor had set in. Aside from rolling, abnormally intense peristalsis rarely plays a rôle in the production of displacements, and if at all, it does so preferably in displacements of the small intestine or small colon.

Torsion of the left portion of the colon (torsio coli) in horses may be brought about in a double manner. When the tolerably long left portions of the colon are well filled and the horse turns rapidly from one side to the other, it may occur that the left portions are not able to follow rapidly the quickly moving right portions. If now the horse, when lying on one side, turns to the other (Jelkmann), or rolls, no matter for what reason (colicky pains), or if it is thrown and turned (Mayer), then displacement may take place in the manner indicated. In such cases the left portions represent the fulcrum, around which the right portions turn, which are fastened to the upper abdominal wall.

In the majority of cases the divisions of the colon show an opposite behavior when torsion occurs. The right or the transverse divisions form the fulcrum, around which the remaining free divisions turn around their axis, either towards one side or the other. In reckless lying down or throwing down, in rolling, in rapid gait, or in jumping, it may likewise occur that the free and quite heavy left divisions of the colon may make swinging or turning motions along the abdominal wall, either to the right or to the left, particularly if they are not well supported by other intestines. The upper division may sink down, especially in its terminal portion which is more or less filled with voluminous and firm contents, and pull the other divisions with it.

Intense and convulsive peristaltic contractions (dysperistalsis), which play an important rôle, according to the belief of Forssell, cannot, directly, bring about torsion of the colon, in the opinion of the authors, because the connected portions of the colon must remain together even if the contractions are irregular. It could not be well explained how the contracting division could get beneath another, quiescent division or how the several positions of the colon could be displaced among each other in consequence of abnormally strong contractions. Convulsive contractions play a predisposing rôle in that they excite colicky pains and cause the animals to throw themselves or roll, and these movements then may cause torsion. In this respect disturbance of circulation caused by thrombosis of the mesenteric arteries, acts like other colicky affections with violent pain.

Predisposing causes always play an important part in con-

nection with the immediate exciting causes. As such may be mentioned heavy filling of the left divisions, especially of the pelvic flexure, with fecal masses, dry masses of feces, sand or calculi, while the transverse divisions are filled less completely; also the stronger filling of the right upper division and, according to Forssell, a stronger filling of the left lower division, the presence of a tumor in the wall of the intestines; a minor degree of filling of the other portions of the bowel; also probably, the variable condition of the stomach as to a greater or a lesser degree of filling and a relaxation of the abdominal wall and according to Joest, the displacement of the apex of the cecum between the abdominal wall and the left lower layer of the large colon; relaxation of the abdominal wall. Forssell also called attention to it that a predisposing cause is furnished in some horses by the comparatively large mesentery between the divisions at the pelvic flexure.

Torsion of the cecum around its long axis is very rare, and it is probably also caused by the mechanical influences pointed out above. Hausotter observed in 6 days 7 cases of torsion of the cecum through 180° in horses which had taken up small quantities of sand and among which several cases of spasmodic colic had occurred a few days previously as a result of bad weather conditions. Schaldemose described a torsion that had turned 1½ times. The shortness of the cecum when compared with the colon and the position of the former, fully explain why displacements of the cecum should be so rare.

Volvulus and torsion of the small intestine or of the **small colon** have a much more complicated mechanism. Torsion may be brought about by a loop of intestine with its mesentery becoming twisted around another loop, the latter with its mesentery forming the axis (volvulus, formation of a nod, volvulus nodosus), or by a longer loop turning around its own mesentery (axial torsion, volvulus mesenterialis). In some cases the whole small intestine of the horse, or the whole colon in the hog may be twisted around the larger omentum with its mesentery, particularly if the body performs very abrupt motions, if the animal throws itself around or is rolled, as occurs frequently in the unloading of hogs and calves. The production of torsion is favored if the intestines contain an abundance of liquid or dry feces or sand (Glage), or if they are the seat of a tumor. However, total or even partial torsion of the mesentery is quite rare.

Especially in horses one observes more frequently entangling or volvulus of the small intestines alone; loops of small intestines are very rarely twisted around the colon or cecum or around the small colon, while loops of the small colon rarely become twisted themselves. In many of these cases, reckless, extensive motions, throwing down, rolling, jumping, etc., cause loops of intestine to become twisted around others.

One may also consider another mode of production. Especially in the horse, loops of small intestine often cross each other in several places under perfectly normal conditions; this arrangement becomes more marked in increased peristalsis, and loops of intestine with a longer mesentery can then move to distant places in the abdominal cavity and below the heavy loops of the large intestine. If the small intestine or the small colon have changed their position in this direction, it may easily happen that the mesenteric veins of the particular loop become compressed to such an extent, that a certain degree of congestion and swelling of the intestinal wall occurs. More active movements of the intestines may now include further loops into the displacement, particularly if neighboring loops cannot get out of the way in consequence of tense filling; this will increase the pressure upon the displaced loops and with it the venous congestion (Wilms). According to Frese, some portions of the small intestines may, as the result of abundant local gas formation or interference with the peristaltic movements, swell up like a balloon and later become twisted as the result of movements of the contents.

Predisposing causes also play a rôle in the production of this form of volvulus or torsion, and as such must be mentioned: Greater filling or greater weight of individual loops of intestine (impaction of feces, circumscribed meteorism, tumor, calculi, parasites), relaxed abdominal walls, and adhesions between individual parts of intestines.

Kinking of the intestines (turn around the transverse diameter) is rare. It occurs most frequently after loops of intestine have become adherent to neighboring organs or to the abdominal wall. However, the small intestine or the small colon will simply be narrowed without the production of disturbances of circulation and its sequelæ; but the stenosis may finally lead to complete closure as in a case of Avérous in a goat. Kinking of the left divisions of the colon (flexio coli) or to the cecum (flexio cœci) occurs occasionally in the horse without preliminary adhesions; it is then undoubtedly due to the enumerated mechanical factors. It is accompanied by rapid development of congestion, because the vessels of these parts of the intestine are included in the kinking. Kinking of the apex of the cecum, with anemia and necrosis of this part (Johne) has been observed a number of times. Also a partial kinking of the wall of the large colon is possible.

Predisposition: The great length of the mesentery of the small intestine in the horse, the fact that most of the colon lies free in the abdominal cavity of this species of animals, the further factor that horses are used for work of various kinds explain the frequency of volvulus and torsion in equines compared with its rarity in other animals. Relaxation of the abdominal

walls and more voluminous feed are the reasons why torsion of the large intestine is more common in heavy horses than in lighter breeds under similar conditions.

Pathogenesis. The facts as to pathogenesis are on the whole similar to those which prevail in internal strangulation (see page 275). The main difference between internal strangulation on the one hand and volvulus and torsion on the other, lies in the fact that the intestine is not strangulated by another firm tissue in the latter condition, but by other loops of intestine.

Anatomical Changes. In torsion of divisions of the colon, one finds the divisions distal from the twisted place distended, dark purplish to blackish red, sometimes spotted with hemorrhages; the mesentery of the two superimposed divisions infiltrated with blood, often gelatinous, the veins in the intestinal wall strongly filled. The latter appears more or less thickened, it is easily torn and is infiltrated with a reddish, serous fluid; the mucosa is blackish red, thrown into folds, pendulous, here and there necrotic. The bowel contains an abundance of hemorrhagic fluid or pasty feces. The congestion is either sharply defined towards the place of constriction or an anemic strip may completely encircle the whole intestine or may be distinct only on two opposite places of the periphery. Exceptionally the congestion may not be very well marked, both on the serosa and the mucosa.

The direction of the torsion may be from right to left, or vice versa, and it may be determined by the relation of the two left divisions of the colon or by the direction of the longitudinal bands. Twists to the right appear to be somewhat more numerous than twists to the left, although in twenty-six cases of torsion Wall saw fifteen to the left and only eleven to the right.

The degree of torsion is very variable. Usually one finds one-half or one entire twist, but several twists are by no means very rare.

The site of torsion (place of twisting, torsion-stricture) is usually found in the transverse colon (diaphragmatic flexure), but, in consequence of falling down of the proximal parts in the region of the right divisions, it may be displaced towards the cecum or to the beginning of the small colon (place of torsion at the root of the colon) and then the cecum may occasionally also be twisted. (Wall, Berlin report 1900-1901). Longitudinal torsion confined to the pelvic flexure has only been described in one case (Berlin report 1901-1902); it is, therefore, a great rarity. The place of torsion is also rarely found in the left divisions (Fig. 23).

Wall gives the following figures for twenty-six collected cases: Fifteen times place of torsion right at the cecum, seven times in the transverse colon, three times in the left divisions, once at the basis of the cecum, so that the latter had also been

twisted; it is possible that the place of torsion is more frequently at the cecum than is usually supposed to be the case, since the finding of the place of torsion in this region is coupled with great difficulties and hence the place may easily be over looked. In torsion at this place it may appear that only the upper division sinks beside or below the ventral division to become strangulated at its junction with the small colon, without changing its position (authors' observation).

In **kinking of the large intestine** the lumen (just like in kinking of any other tubular organ) becomes narrowed or obliterated by the inward projection of the wall, and the parts beyond the kink show signs of intense congestion. In kinking of the apex of the cecum the shut off portion usually becomes anemic and necrotic.

In **torsion of the mesentery of other portions of the intestine**, the mesentery itself is twisted into a cord or there is a volvulus when one portion of the intestines has wound around another portion, and strangulated it like a ring (fig. 24). Parts of intestines strangulated in this manner appear enormously distended and are dark red in color. Otto found in a dog a torsion of the entire large intestine up to the cecum.

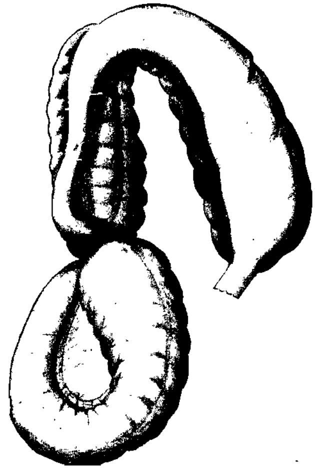


Fig. 23. Torsion of the left divisions of the colon towards the right in a horse suffering also from volvulus of the small intestine. (As a rule torsion occurs more anteriorly.)

The peritoneal cavity contains, in any form of volvulus or torsion, a bloody serous fluid; sometimes circumscribed or diffuse peritonitis is present, occasionally intestinal rupture. Besides there may be found indications of sepsis or sapremic intoxication, particularly in torsion, about the long axis of the large colon, and frequently signs indicative of death from suffocation.

The differential diagnosis of torsion or volvulus must consider hemorrhagic infarction of the intestinal wall in thrombosis or embolism of the mesenteric arteries in those rare cases when on postmortem examination the displacements are not very noticeable or are overlooked entirely. However, a careful examination of the blood vessels, as to the size and position of any thrombi or emboli present, and careful inspection will prevent errors. The sharp demarcation of hemorrhagic infiltration in a straight or curved line is without significance, because it is likewise seen in thrombotic or embolic infarction (see page 436). It appears doubtful whether one is justified, as Wall claims, to attribute congestion in the colon, in the absence of

any torsion, to a stricture which has existed during life, but which has become relieved spontaneously before the animal died.

Postmortem displacement of the intestines can be distinguished from those formed during life, because in the former case signs of congestion are absent.

Symptoms. External symptoms of torsion are in general identical in various species of animals with those of internal strangulation of the intestine (see page 278.) A decided difference exists in the main in the fact that volvulus and torsion generally lead to a considerable increase of the abdominal cir-

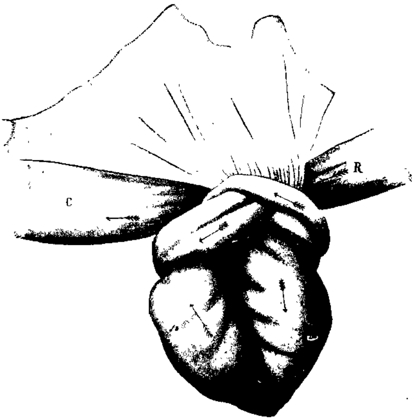


Fig. 24. Volvulus of the small colon. *C*, small colon, *R*, rectum (according to Mollereau).

cumference, because the large intestine or a considerable portion of the small intestine are involved, hence a good deal of bloating will occur.

In the case of **torsion of the colon** in the horse, rectal examination will show a high degree of meteorism. One finds both left divisions, particularly the lower one, considerably distended and reaching up to the upper abdominal wall, so that sometimes it is possible only with the greatest difficulty to force the hand between the bloated bowel and the upper abdominal wall. Firm feces cannot be felt through the much distended and very tense elastic wall. If, however, the patients are examined at once after the onset of the affection or if the latter progresses exceptionally slowly, extensive bloating may be missed for some time. The other portions of the intestines first remain of normal size, but may often be displaced by the colon out of their proper

place (especially the cecum). After a longer duration of the disease the other portions of the intestine likewise become bloated, but never to such an extent as the colon.

The position of the colon either remains normal or one finds the two left divisions displaced towards the median plane of the abdominal cavity, or even towards the base of the cecum. The pelvic flexure is directed either backward or towards one side, or towards the thorax, and the course of the divisions of the colon is either straight or towards one side, or curved in a bow. The mutual relation of the left divisions of the colon remains either normal (in one complete torsion) or their relation has changed so that the smooth upper division, becoming larger towards the thorax, is situated either towards the right or left, or below the much thicker lower division, and presents bands and sacculations. If the pelvic flexure is displaced toward the front, it can be palpated per rectum only in small horses. If, however, the upper division is situated more or less laterally from the lower division, the pelvic flexure is found more or less horizontal on the lower abdominal wall instead of vertical as found under normal conditions. The two free bands of the lower division (one lateral and one median longitudinal band) show the same course as the lower division of the colon itself, but they are also influenced by the position of the pelvic flexure. A spiral course of the longitudinal bands to the right or left can be ascertained particularly in those cases where the spiral formed in twisting reached into the neighborhood of the pelvic flexure, or where the latter has been bent towards the front, otherwise the bands show a normal course. Pulling on the colon or on its longitudinal bands frequently causes pain to the patient. The point of torsion, where the divisions of the colon are twisted into a spiral, tough cord, in a circumscribed place, can only be recognized in those very rare cases where torsion has occurred in the posterior half of the left division of the colon (Fig. 23).

Kinking of the colon or of the **cecum** may be assumed when the intestinal wall, in some place, forms a sharp angle, and when the bent portion of the intestine is strongly bloated. The place of kinking must, of course, be at a point accessible to rectal exploration, otherwise it cannot be recognized.

Torsion of the cecum can probably be often diagnosticated by rectal exploration as was shown in a case which the authors observed.

Torsion or volvulus of the small colon (often called the abdominal portion of the rectum) can always be ascertained by rectal examination. If a portion of the small colon, situated very near the rectum, has been closed, the introduction of the hand through the narrowed place is impossible, perhaps a finger may be pressed through. One finds the intestinal wall folded

and tender in front of the impediment; bloated loops of intestines can be felt through the wall of the rectum. If torsion has occurred more towards the front, the findings are similar to those in strangulation of the small colon (see page 280), with this difference, that in volvulus with the small intestine the strangulated small colon is surrounded by bloated loops of small intestine.

Formation of knots and partial mesenteric torsion of the small intestine may in some cases be ascertained directly by the observation that loops of intestine, which are bloated to the utmost and very tense, suddenly pass over into a folded cord, which, on pressure or on pulling, proves to be tender. Furthermore, a velvety to gelatinous character of the thickened intestinal wall and of the mesentery and an intense engorgement of the veins may be noted.

In horses that are not too large, total torsion of the small intestine around the root of the mesentery may be recognized by finding a folded, thick cord, in a vertical plane, supposed to pass through the middle of the left kidney, immediately below the vertebral column, which leads towards the right or left and is connected with a number of very tense loops of small intestine. In some cases one may also find loops of small intestine wound around the caudal portion of the left division of the colon, or around the upper portion of the cecum. In a considerable proportion of cases one cannot find the seat of a torsion or a volvulus of the small intestine, but can only ascertain very great distension and eventually tenderness of some loops, while the rest of the small and the large intestine are normal (circumscribed meteorism). Only rarely does rectal examination furnish a completely negative result, namely, when the displacement has occurred in the anterior parts of the abdominal cavity and when only a short loop is involved.

Rectal examination, or palpation of the abdomen, may reveal conditions similar to those of internal strangulation (see pages 280 and 281) in **cattle, hog and carnivora.** Probst saw the case of a dog with torsion of the rectum and with bloody feces (probably the torsion was originally not complete in this case). The authors saw a similar case in a horse with torsion of the rectum.

Course. The clinical picture sometimes develops during the course of other colicky affectious, but, as a rule, it has a very sudden onset. As in displacements of the intestines in general, either intense symptoms of restlessness occur at once or they increase gradually in intensity. The disease is of short duration, since death occurs rapidly, particularly in displacements of the small intestine, sometimes after a few hours, usually towards the end of the first or during the next day. With torsion of the large intestines, particularly with torsion of the

small colon, horses may live two or three days. In other domestic animals the course is similar to that in strangulation of the intestines.

The possibility of spontaneous recovery cannot be excluded a priori; but spontaneous reduction may occur only in displacements of smaller sections of the intestines and only in the beginning of the disease, that is, as long as bloating in the affected portion does not yet exist, which would, of course, make a spontaneous recovery impossible. (Frese found on post-mortem in 5 cases that a torsion of the colon had recently become spontaneously rectified; a similar condition was found in several of the author's own cases.)

Diagnosis. Torsion and volvulus can only be diagnosticated beyond doubt if rectal examination in large animals, or abdominal palpation in smaller ones, reveals the place of twisting or entanglement. Torsion of the colon can be diagnosed almost with certainty by the finding of a fleshy thickening of the wall of the left colon and mesocolon together with other manifestations of intestinal displacement, in which case only infarct of the intestinal wall through thrombus or embolus must be eliminated. The other symptoms, by themselves, permit only a probable diagnosis which nevertheless may come very close to certainty if the clinician has had much experience.

Corresponding to the very frequent localization of the torsion in the transverse colon, and of the right colon, the torsion of the colon can be diagnosed only as probable. Jelkmann gives as a characteristic sign that two tense strings can be palpated in the plane of the 4th lumbar vertebra; according to Moeller the spiral course of the two tense strings of the left lower colon is of importance. According to Forssell the relation of the left portions of the colon, and the course of the longitudinal bands is of deciding value. Regarding these points, the fact is not to be neglected that in primary and secondary fecal accumulation or distension, the position of the colon is often changed (Figs. and 26), the left upper portion being usually to the right, less frequently beside or beneath the lower portion, whereby the normal position often is assumed only in the portion of the transverse colon in which the physiological position cannot be determined; hence a true torsion is often simulated. In this case and also in the turns of the pelvic curvature which look to the right or oro-ventrally, the bands of the lower portion usually take a more or less spiral course and the left portions may bulge in toward each other without leading to a noticeable diminution of the intestinal lumen or to stasis of the blood.

Equally uncertain is the determination of the direction in which torsion of the colon has taken place. The direction of the torsion is determined by Möller from the course of the longitudinal bands. In torsion to the right, the band of the lower divisions lead from in front towards the back and to the right; in torsion to the left in the opposite direction. Forssell gives the following rules:

1. If the left upper division lies to the right of the lower division and if it passes here obliquely forward, upward and to the left, then

we are dealing with a half twist towards the right. If the left upper division passes obliquely forward and downward and to the left, then we have a twist to the left of more than 90°.

2. If one feels the left upper division to the left of the lower division and if it leads obliquely forward and downward and to the right, then we are dealing with torsion to the right of more than 90°; if it leads obliquely forward and upward and to the right, then we are dealing with a half torsion to the left.

3. In those rare cases where the left lower division completely covers the upper division, the spiral course of the band and indentations of the left lower division which may be present are the deciding factors.

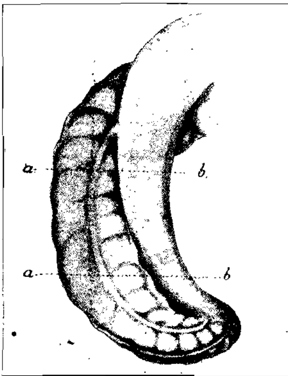


Fig. 25. Incomplete torsion of the colon in a horse. The pelvic flexure is displaced to the right, the left superior division lies at *a*, *b*, beside and under, whereas at *a*, *b*, it lies beside and over the inferior division.

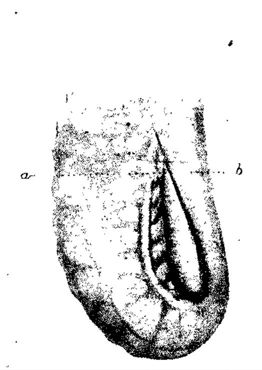


Fig. 26. Incomplete torsion of the colon in a horse. The right superior division lies beside and under the left inferior division; it passes, however, at *a*, *b*, into the normal position.

The diagnostic value of these signs is limited because they are observed also in pseudo torsions and the left portions with their bands may run in a straight direction, if the point of torsion is in the transverse colon or in the region of the right portions. If a pseudo torsion develops in such a case, it may even simulate the opposite direction of torsion.

Concerning the details of diagnosis, including differential diagnosis, the reader is referred to the subject of internal strangulation (see page 283). Even where rectal examination or external abdominal palpation give information, the affection cannot always be differentiated from internal strangulation, but this is of no great importance, since the therapeutic procedures are the same in both cases.

Treatment. The same principles as those laid down for the treatment of internal strangulation (see page 285) govern on the whole the treatment of torsion or volvulus. However, the very nature of these affections brings it about that, except in a case of torsion of the colon, reposition from the rectum is even less frequently possible than in internal strangulation.

In torsion of the colon one should always roll the horses in the direction of the torsion whether it be diagnosticated positively or only suspected. Forssell has recently had some good results with this procedure (17 good results in 18 treated horses). The value of this method cannot be determined accurately, because of the uncertainty in excluding the pseudo displacements from other diseases associated with colic, which are less dangerous. Other observers (Behrens, Hummerich, Marek) have not been able to obtain these splendid results; Hummerich and Kolcher have treated many cases of primary intestinal meteorism and of fecal accumulation in this manner with good results. The best chances for reposition of the bowel, by rolling, exist if the seat of the torsion is at the point where the transverse colon passes into the left portion, or in the region of the left colon.

Relief of torsion of the colon by rolling, according to Forssell, is brought about as follows: According to the Stuttgart method the horse is best placed on the side corresponding to the twist of torsion, that is, if torsion is towards the right, the horse is placed on the right side. Before this is done the bloated colon must have been punctured either through the left abdominal wall or through the rectum (see page 218). Then the arm of the operator is introduced into the rectum; he next tries to fix the pelvic flexure with his fingers (in mares one may make an incision into the vagina for this purpose); indeed, according to Forssell, the intestine becomes fixed by its own weight. Then the horse is rolled on its back and abdomen till the intestine gets into its proper position. (A horse had to be rolled nine times in a case of Forssell before replacement occurred.) To prevent rupture of the rectum, the fixation with the fingers must be interrupted from time to time. The reposition of the bowel is usually announced by the expulsion of intestinal gases, although sometimes flatus does not immediately occur. The restlessness persists for a few hours if the rolling was performed a few hours after the onset of the disease; if, however, the horses have been sick for fifteen or twenty hours, they become quiet almost immediately after reposition. The after treatment consists in starvation or in very careful feeding for several days. Steinbrüch had a horse turned on the back, and then had the posterior portion of the body raised, with a pulley, proceeding gradually and lifting the rear portion of the body three feet high for the space of forty-five minutes; during this time 36 quarts of lukewarm water were allowed to run into the rectum.

The **correction through the rectum of torsion of the colon,** according to Jelkmann and Möller, is performed on the standing animal after a preliminary puncture of the intestine. The posterior abdominal portion of the animal is first elevated, the hand is then introduced into the rectum, and guided towards the left abdominal wall. The hand then tries to push forward and inward the accessible loops of the small colon and those divisions of the colon which are above the former; if the median line has never been reached the hand is guided upward, whereupon, in favorable cases, the colon glides into the place of the loops of the small colon which have been pushed aside; in this manner the torsion may become relieved.

This procedure was recommended by Jelkmann and is indicated only in torsion towards the left. Möller proceeds in another manner. He pushes with the hand, introduced into the rectum, the upper division, which is twisted towards the left, upward and at the same time the lower division downward and to the left.

The experiences of Larsen, Keutzer, Forssell, Straube, however, and the observation of the authors show that, even with a good deal of force and persistency, torsion cannot be corrected because the colon is such a heavy body that it cannot

easily be moved in the closed abdominal cavity where neighboring loops of intestines at once occupy every space that might be made free. Forssell also calls attention to the ever present danger of rupture of the rectum. Jelkmann, Möller, Malkmus, Zippel and others have had good results in those cases which belonged to the type of cases recommended for their procedures by Jelkmann and Möller.

Kinking of the pelvic flexure can be corrected from the rectum by pulling towards the pelvis (Diem). Sigl has relieved a case of torsion of the small colon by the injection of water into the section situated in front of the impediment. Levens relieved a torsion of the small colon by countertorsion, while Meads believes that he relieved a suspected volvulus by introducing both hands into the rectum, grasping the point of torsion with the right hand and holding it in place while he pushed the colon over with the left hand.

When a reliable diagnosis has been made laparotomy is usually the only indicated procedure except in cases of torsion and kinking of the large intestine. Aside from its danger to life, however, even this procedure cannot always produce the desired result, as is evident from the nature of the disease itself. The experiences of the authors show that volvulus or extensive torsion of the mesentery cannot be cured even by this operative procedure. In torsion of the colon, laparotomy with subsequent rolling may be indicated (Forssell). Concerning the indications for laparotomy the reader is referred to the subject of intestinal strangulation (see page 286).

In a case in a horse where loops of the small intestines had become twisted around the small colon Meschkow was able to replace the displaced loops after opening up the right flank; recovery took place. Sigl had a similarly good result in volvulus of the small colon. Audebert replaced divisions of the colon, alleged to have been displaced, with the hand introduced through an incision into the wall of the vagina. Piósz & Marek were unable in two cases to reduce a mesenteric torsion of almost all of the small intestine after laparotomy. Ries performed laparotomy in cattle in two cases; he succeeded once in correcting a displacement, while Joehnk succeeded in correcting invagination and torsion which were present simultaneously. Grasby and Reynolds relieved a case of volvulus in a yearling cow by laparotomy in the region of the flank.

Since reposition of twisted divisions of the colon might be brought about spontaneously by rolling, moderate rolling of the patients may be permitted. In these cases, and in torsion or volvulus in general, the pain should be relieved by narcotics (morphine, chloral hydrate). Intestinal puncture (see page 218) is indicated, without any reference to further treatment, whenever there is intense bloating; some cases of intestinal flexure (see page 293) or of other displacements of the large intestine may occasionally assume their normal posture after the escape of gases. The use of laxatives is contraindicated. The favorable results claimed by Oeller and Ohler can not be judged objectively on account of the insufficient clinical diagnosis in these cases.

26. Intestinal Invagination. Intussusception. Invaginatio intestini.

By intussusception one understands a stenosis or occlusion of the intestinal lumen which occurs suddenly and is caused by the invagination of one section of intestine into a neighboring

part, followed by a venous congestion in the invaginated portion.

Occurrence. Invagination is found most frequently (but even then as a rare occurrence) in cattle and somewhat more rarely in dogs, very rarely in the horse and in the hog. Walker, in South Africa, observed numerous cases of invagination (Reckziekte, Knopdarm) in sheep over 15 months old. On some farms from 5 to 10% of the sheep died from this affliction. Also Raupp saw invagination in sheep. Sometimes it is observed in fowls (Kitt, Johne, Klee, Eber). In general, young animals are somewhat more frequently affected.

Among the horses of the Prussian army in the years 1892-1912 there occurred in 98,249 cases of colic 119 cases of invagination (0.12%); at the Berlin Clinic in the years 1897-1910 in 10,893 cases of colic there were 13 cases (0.12%) of invagination; at the Dresden Clinic in 3,901 cases of colic there were 5 cases (0.13%) of invagination, while at the Budapest Clinic invagination was observed in 0.17 to 0.31% of all colic cases.

Etiology. Permanent invagination occurs only in abnormally active and energetic peristalsis. Hence all stimuli which increase peristalsis may bring about invagination indirectly.

The peristalsis may be increased in all or in certain parts of the intestines, and become convulsive in sections, after a cold, after the ingestion of ice cold water or cold feed (frosted grass, frozen bulbs), (Wagenheuser saw the disease after ingestion of frosted grass simultaneously in 7 colts), or accompanying intestinal catarrh or enteritis, intestinal parasites, foreign bodies in the intestinal tract, tumors on the intestinal wall and colics in general. In the observations of Walker the numerous worm nodules due to larvæ of **Œsophagostomum** occurring especially at the point of invagination might have played an important rôle, as the rough and stiff intestinal wall prevented the rectification of invagination, which occurs also during normal peristalsis over short distances.

It is not impossible that in certain cases a section of intestines, which is just contracting, may become invaginated into a neighboring piece by a sudden great increase of intraabdominal pressure. This may explain those cases in which horses suddenly fall sick in jumping, and cattle in running about, or during parturition, or in drawing a load uphill. Even in these cases, however, the peristalsis which has become more intense, is probably the immediate causative factor.

As predisposing causes must be considered the firmer attachment by a short mesentery of some portions of the intestine, a heavy tumor, adhesions, occasionally also bloating.

The seat of an invagination is most frequently the small intestine, since it is especially subject to stronger contractions; more rarely the ileum becomes invaginated into the cecum, exceptionally into the colon (Johne); in horses, cattle and dogs, the apex of the cecum into its body; in horses and exceptionally also in dogs the cecum into the colon, rarely the

small colon into itself or into the rectum. There has also been observed invagination of the duodenum into the stomach (H. Bouley in a horse, and Peuch and Cadéac each in one dog). This occurrence must have been due to antiperistalsis which may also cause invagination in other portions of the intestinal tract into a section situated towards the stomach. According to Joehnk, the place of invagination is, in cattle, most frequently the point where the jejunum passes into the ileum, fairly frequently also in the loop of the last portion of the colon, but very exceptionally in the region of the colon loops which are united firmly. Walker always found the invagination of sheep in the ileum 23 to 164 cm. in front of the ileocecal valve.

Pathogenesis. In accordance with Nothnagel's experiments, brief and transitory invaginations of the intestine may occur physiologically, during normal peristalsis in such a manner that the portion of the intestine, which has become thinner and longer because of the contraction of the circular muscles, is forced into the adjoining distal portion, which is dilated because of contraction of the longitudinal muscles, provided the latter is fixed more or less or is less movable, or if the proximal, elongated part of the intestine is prevented from rolling aside by an unyielding abdominal organ. This invagination is restored the next moment because of the relaxation of the circular muscles, as only a short bit of bowel has entered and the muscular contractions are of very brief duration. A lasting, permanent, pathological invagination occurs only if the peristalsis is increased morbidly, and if the contractions are prolonged; in this case elongated portions of intestine can be pushed into adjoining ones, and if they remain in such a position for some time, a moderate stasis of the blood may develop and lead to swelling of the intestinal wall by which the solution of the invagination is prevented, after the muscles have become relaxed.

In a similar manner the agonal invagination is produced under the influence of an increased peristalsis because of the greater carbonic acid content of the blood, during the agony and immediately after death. In these cases, however, the usual results fail to materialize because the circulation ceases after death.

According to the paralytic theory, which still has many adherents, the invagination occurs in such a manner that single portions of the intestine are paralyzed under certain conditions, while the proximal portions are still in lively peristaltic motion and therefore slip into the immovable or relaxed portions of intestine. This view is contradicted by the different frequency of invagination in different species, and also its greater frequency in young animals in which comparatively slight causes may incite a brisk peristalsis. Moreover, it would not be possible that this displacement of the intestine is least frequent in horses, because in them there would often be an opportunity on account of frequent partial paralysis of the intestines. Finally it would be inexplicable that often very long parts of intestine become invaginated.

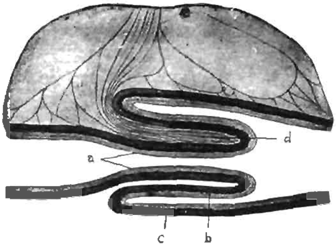


Fig. 27. Diagrammatic representation of invagination. *a*, internal, *b*, external portion of the intussusceptum. *d*, its mesentery, *c*, intussuscipiens.

Wherever invagination occurs, the intussuscepting portion will, of necessity, draw along its mesentery with the blood vessels of the latter (Fig. 27). The intussuscepting portion and its mesentery will be subjected to a certain amount of pressure, depending upon the caliber of the intussuscepted section; this will cause congestion, swelling and subsequently a hemorrhagic serous infiltration of the drawn-in portion of the intestine and its mesentery. The extravasating hemorrhagic fluid gets into the lumen, also between the invaginating and the invaginated portions, and also to a small extent into the free peritoneal cavity at the place of entrance of the invaginating portion.

Passive congestion, which increases rapidly in the intussuscepted portion, causes marked convulsive contractions with colicky pains, and the former may cause an increase of the portion invaginated towards the anus. This is favored by the fact that while the circular fibers of the intussuscepted portion contract, the longitudinal fibers of the intussuscepting section likewise contract. If the place of invagination is somewhat fixed, the latter section will slip over the former. Contraction of the circular fibers of the invaginating portion of the intestine may cause an increase in the invaginated portion, because the contracted elongated bowel may, at its anterior end, slip further over the invaginated bowel. These factors may sometimes bring it about that the invaginated intestines may be several meters long. The increasing contractions of the invaginated intestine with its mesentery causes continuous pain which, as usually in displacements, becomes augmented by external or by increased intraabdominal pressure. However, the pain is, as a rule, not very intense.

The lumen of the invaginated bowel is considerably diminished from the start, and it may not rarely become entirely obstructed in consequence of swelling of the intussuscepted portion, together with its mesentery. The larger the intussuscepted bowel, the less the lumen of the intussuscepting portion will be diminished. Diminution or obstruction of the intestinal lumen excites more active, later on convulsive contractions in the intestines situated towards the stomach; these contractions

Fig. 28. Invagination in a horse.

are moderately painful. The peristalsis of the sections situated towards the anus only ceases when the invaginated portion has lost its contractility; this occurs in consequence of intense infiltration, necrosis, secondary peritonitis, or excessive compression; usually, however, only after a number of hours and even days. As long as the invaginated bowel moves, the peristalsis progresses towards the anus, even if complete obliteration supervenes eventually, because the strangulation is not so severe that the peristalsis may not progress from the invaginating toward the invaginated portion. These contractions may be sluggish; they continue for some time or even until the end, and propel the serous, hemorrhagic fluid, that is extravasated into the lumen, towards the rectum.

The nutritive disturbances which are caused by the congestion of the intestinal walls may be very variable in degree, according to the nature of the affection, and lead to similar consequences as they are found in internal strangulation; the immediately affected portions of the peritoneum are, however, less endangered (see page 279).

In those very rare cases in which the patients remain alive for a long time, the invaginated necrotic portion of intestine may occasionally become detached; if this does not occur and if there has not been complete stenosis, the latter will then occur.

Anatomical Changes. The two invaginated portions of intestines form a curved, screwlike, or tortuous cylinder (Figs. 28 and 29) which is firm, sausagelike in consistency or fluctuating. It is usually several centimeters long and occasionally measures several meters long. In the latter case one can feel a sausagelike body in the affected part. The bowel looks normal externally, or purplish to dark bluish-red, because the folding of the mesentery also compresses the veins of the outer portion. At the end of the cylinder one sees the place of entrance of the

narrowed portion of bowel with its mesentery (intussusceptum) into the most external layer of the wall composed of three layers (*intussuscipiens*, see Fig. 27). Wall and mesentery of the intussusceptum exhibit intense venous congestion, and at the point where the intussusceptum folds back the apex can be recognized. Where the ingoing and outcoming portions of the intussusceptum are in contact with each other, fibrinous peritonitis is soon developed, hence the pieces of intestines can be drawn apart only with difficulty or not at all. An invagination which has been formed during the agonal stage can easily be reduced and does not show congestive or inflammatory changes. Invagination is, as a rule, single, but it may be double, triple or quadruple. Sometimes invaginations are found in several

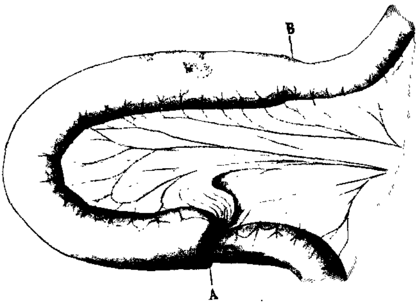


Fig. 29. Invagination of the small intestine in a dog. A, Place of entrance; B, conical end of the invaginated bowel.

places. Finally, one may find the evidences of a general peritonitis.

Symptoms. In horses invagination produces on the whole the same clinical picture as internal strangulation (see page 272). In invagination of the cecum into itself or into the colon defecation is not entirely suppressed, or at least not continuously; the animal may even survive; but after the violent symptoms have disappeared those of intestinal stenosis remain behind (see page 249).

Rectal examination often shows important differences between invagination and other intestinal displacements. Circumscribed meteorism is absent until the end, except in protracted cases, when all the intestines become moderately bloated. In invagination of the small intestine into itself, the hand introduced into the rectum feels a thick, elastic-firm, painful, sausage-

like body which may be wound up like a snail, of the consistency of meat; at other times a more or less fluctuating loop of intestine in which, on firmer pressure, a sausagelike body may be felt. Sometimes only the place of entrance can be reached, where the intestine becomes thinned out to a cord and disappears in a thick fleshy ring. In invagination of the ileum into the cecum, one feels in the head of the cecum a body as large as a wrist, somewhat tender, movable and of tough-elastic consistency (Klett). Occasionally invagination of the cecum into itself may be felt, or of the cecum into the colon.

In those rare cases where invagination did not occur towards the front and where peristalsis did not cease in the portions towards the anus soft hemorrhagic feces are seen.

In **cattle** the clinical picture is also quite similar to that found in internal strangulation (see page 282). Kolb noticed in one case stretching in urinating in the manner seen in male horses. Joehnk observed at first convulsive twitchings, and the symptoms of febrile hemorrhagic gastroenteritis. In contrast to other intestinal displacements, the intestinal discharges are often bloody in that either the feces are mixed with blood, having become of thinner consistency, or then a bloody, glary slime is expressed with much exertion, or fibrin-like masses mixed with blood. According to Joehnk, the pressing with bloody slime is observed only while the animals are standing and can easily be made to appear if the animals are made to stand up.

Rectal examination gives similar results as that in horses but in cattle the invaginated part of intestine usually can be palpated in the posterior part of the abdominal cavity which is bordered by the rumen and by the right abdominal wall (Joehnk).

In **sheep** Walker did not see intense abdominal pain, but only observed frequent looking around at the abdomen, stretching similar to that of male horses when urinating, thereby bending the back downward, standing or lying motionless, changes in the feces similar to those observed in cattle, sometimes insignificant intestinal hemorrhages, and usually a fatal termination.

In **dogs** the clinical picture of invagination differs from that of internal strangulation only by difficult defecation and bloody fetid feces are observed. The general condition, pulse and respiration of the animals are little or not at all disturbed for one or two days; the appetite is, however, suppressed from the start. Parent saw prolapse of the invaginated bowel at each defecation in a young dog with invagination of the colon into itself. Palpation of the abdomen reveals an elastic-firm, cylindrical, sometimes curved, painful body which can be moved freely in every direction. One can sometimes feel the conical end of the invaginated part and on the other side the turning point of the external tube of the sausagelike body (Fig. 29).

In a case of Hutyra's the emaciated animal showed for a long time obstinate bloating and thin-fluid stools.

Course. After the disappearance of the abdominal pains, which are at first very intense, several hours elapse (in cattle usually six to twelve hours), occasionally several days (particularly in horses), then the animals become quiet, and they die from complications which have developed. In horses several attacks of colic may be observed exceptionally, occurring at brief intervals, while some authors claim that in cattle the disease may run its course without any pain. Pains may, however, be overlooked of course in animals which are not always under observation (at night).

The disease lasts usually six to nine days in cattle and may rarely be prolonged to two weeks, while a short course of only a few days is rare, in sheep 4 to 14 days. In horses invagination of the cecum into itself or into the colon may run a course of six weeks (Panthe) or even of months (Colin); during these long periods the animals present the symptoms of intestinal stenosis.

Recovery is rare, but possible if the invaginated portion becomes cut off at its oral end and is voided with the feces, in cases where adhesions have formed between the serous layers at the entrance of the invagination. Cases of this kind have been reported in horses (Verrier, Rackow, Martin, Hochstein, Perkuhn) and also in cattle. Schantyr observed a recurrence of the invagination in a dog in which the ileum had become invaginated into the jejunum.

Diagnosis. Intestinal invagination may be diagnosticated with certainty by positive findings on rectal examination; in very rare cases a diagnosis may be made later in the course of the disease, on account of the expulsion of pieces of intestines with the feces. Hemorrhagic feces with mucus or even masses of fibrin, also symptoms of intestinal obstruction and signs of general disease can, in spite of an early disappearance of the restlessness, create only a suspicion of invagination; they cannot be relied upon for a definite diagnosis.

On the basis of the results of rectal examination, the affection may be confounded with impaction in the small intestines and also, in cattle, with impaction in the large intestine; impacted intestinal loops feel more doughy or harder, less elastic, nodular, not painful, and often several loops show this condition. One also finds occasionally thick-walled, painful elastic portions of the intestines in membranous enteritis, but the symptoms of intestinal occlusion are then missing. Invagination in cattle cannot always be differentiated from volvulus or from obstruction of the intestines with coagulated blood; this is, however, immaterial as far as further therapeutic procedures are concerned. In dogs the affection might be confounded, after

abdominal palpation, with impaction in the large intestine; but in obstipation we find a soft-firm or then a hard, nodular, cylindrical mass, not movable, not elastic and, as a rule, not painful; defecation does not occur. The affection of invagination in dogs may more easily be confounded with obstruction of the intestines by foreign bodies, so that an exploratory laparotomy alone can decide the diagnosis. In cows the differential diagnosis must exclude torsion of the uterus (Albrecht).

Treatment. The only treatment that promises any success at all is laparotomy. After opening the abdominal cavity (in cattle in the right flank, in dogs and possibly also in foals, in the median line, in horses at a point next to the seat of the invagination) the invaginated intestine is located and drawn out. The loops are then reduced or, if adhesions have formed or there are signs of necrosis, the affected portion of the intestine is resected and the ends of healthy intestine are united by a suture. In separating an invaginated portion it is advisable not merely to pull, but to press at the other end.

The operation was first performed in cattle by Luscan in France and by Meyer in Germany, and good results were obtained in this animal. (Luscan, Meyer, Taccoen, Hafner, Matthieu, Perdan, Guittard, Schmidt, Schiel, Jöhnk and others); Michener cured invagination in young foal; Plósz obtained good results in several dogs by laparotomy made in the median line.

Whether it be possible to free the invaginated intestine with the hand in the rectum in larger animals, or through the intact abdominal wall in smaller animals, is a question which cannot yet be answered.

The use of laxatives is contraindicated; they can only do harm. The injection of water might be tried, but one can expect good results only in certain invaginatious of the small colon or of the rectum.

27. Colic.

The term colic means intestinal pain, according to its etymologic derivation (dolor coli, colica, neuralgia colica, enteralgia, enterodynia). By typical colicky pains one has always meant in human medicine peculiar, cutting, pinching or tearing intestinal pains (Bauchzwicken [German]) which come on suddenly in the form of an attack and disappear again suddenly after a certain period of time. Nothnagel has shown and the authors have become convinced from the results of their experiments and rectal examinations that genuine colicky pains are always dne to convulsive (tetanic) contractions which cause compression of terminal sensory nerves. The experience of veterinary practitioners leaves no doubt that contractions of the muscularis of the stomach have a similar effect. The experience of both human and veterinary medicine has likewise shown that convulsive contractions of the smooth muscle fibers of other organs (bile duct, urethra, urinary bladder) also excite genuine colicky pains.

Convulsive contractions of the stomach and intestines in domestic animals are always secondary in nature and due to a variety of diseases of these organs which may either increase the excitability of these parts (catarrh, inflammation) or which may lead to abnormal intense stimuli (sudden passive hyperemia, mechanical irritation, irritating decomposition products, like carbon-dioxide, methan, fatty acids).

Since colicky pains are only a part of a group of symptoms of various heterogenic affections, it does not appear justified from a pathological standpoint to consider colic as a definite pathologic condition; one might possibly distinguish various varieties of colic, such as catarrhal-, impaction-, gallstone-colics and so forth, as is frequently done by present-day writers.

In considering the advisability, whether the collective designation "colic" should be retained, one must keep in mind the fact that horses do not suffer only from true colic, but that they are subject to various diseases of the abdominal organs or of those in their neighborhood (peritoneum, liver, kidneys, pelvis of the kidneys, urinary bladder, sometimes also of the pleura) with inflammatory, dull continuous pain. Such pains are also experienced in the course of certain affections of the esophagus, and so are certain disagreeable sensations in the region of the rectum, which are due to parasites, continued hunger, great exhaustion. One can, therefore, not even always determine from the behavior of the horse, whether the animal really suffers from true colic, and this can only be determined after the underlying affection has been ascertained.

If, however, genuine colicky pain is only a part of the clinical picture of heterogenous diseases and if socalled colicky symptoms are not always true colicky pains, it is obvious that the collective term "colic" can no longer be justified from a scientific standpoint.

Not even from a practical standpoint does the use of the term colic appear well founded, because it might lead the veterinary practitioner into the belief that "colic" was a unity which is always to be treated according to the same principles. It is possible to-day to differentiate many of the diseases belonging to this group on the basis of a proper examination of the patient. Such a differential diagnosis can now be made more frequently than the differentiation of various diseases of other groups, which yet are not united under collective names. For the sake of convenience one might retain a collective name for the most heterogenous affections. It would undoubtedly be more convenient to treat a patient by routine for colic without an exhaustive tedious examination and without taking into consideration the real requirements of the case, but this procedure will surely do harm in many cases, where treatment would be successful if it were administered as indicated by the particular underlying affection.

One should, therefore, only speak of "colicky affections,"

"coliclike affections" of the stomach, intestines, gall ducts, ureters, for the sake of brevity and for descriptive purposes, in the case of diseases which may have either true colicky pains or coliclike pains as one of their symptoms.

After the necessity of separating different forms of colic had become obvious, a number of sub-groups were formed. Diseases of the stomach and intestines which are characterized by abdominal pains and usually also by disturbances of defecation or by suppression of peristalsis have been distinguished as genuine colic, from false and from symptomatic colic. With the name false colic were designated painful affections of the peritoneum, the spleen, the kidneys, the internal organs of generation, with the name symptomatic colic attacks of pains occurring in the course of such diseases as anthrax, purpura hemorrhagica, poisoning, gastro-intestinal inflammations.

If, in closing the discussions of diseases of the stomach and intestines, which may be the cause of symptoms of colic, the authors here add a special chapter devoted to a general description of colicky affections, they do so from various motives. Without such a chapter they could, in the first place, not clearly define what is meant by *genuine colicky pains and differentiate them from other pains which are not of* a true colicky nature, but which manifest themselves in a similar manner. Besides unnecessary repetitions in describing the symptomatology of diseases of the stomach and intestines may be avoided by treating of symptoms of colic in a separate chapter. And it is finally not at all superfluous to treat collectively the differential features of all diseases which may give rise to the symptoms of colic.

Diseases of horses leading to the symptoms of colic are very frequent. This is shown among other observations by the statistics of the Prussian army, showing that three to five per cent of all horses annually become affected with "colic," while the proportion of the total morbidity to "colic" is like 100 to 11-14. In veterinary clinics one-half or even more of all horses brought to the clinic suffer from colic; this very high figure is due to the fact that horses suffering from other diseases which lead to less obvious symptoms than "colic" are not brought to the clinic so frequently.

The mortality of colicky affections varies within wide limits because the underlying diseases are different under different conditions and in various localities. This figure varies between 10 to 17 per cent or perhaps even a little higher; it is, on *an average, about 12 per cent.*

Various circumstances afford the reasons why colicky affections are so common in horses. Horses appear to be more sensitive to pain than other species of animals. It is also part of the nature of the horse to react to disagreeable or painful sensations in a very marked manner. Moderate pains often do not become noticeable at all in other species of animals, although they are felt.

Another cause of the frequency of colicky affections in the horse are the anatomical conditions of the abdominal organs. The stomach is comparatively small, the pylorus is situated between the blind pouch of the stomach and the stomachlike dilatation of the large colon; this predisposes to overloading or dilatation of the stomach. The unusual length of the mesentery of the small intestine and the mostly free arrangement of the

voluminous large intestine of the horse, predispose to certain displacements of the intestine under certain conditions (strangulation, volvulus or torsion).

The physiologic condition of nutrition in horses likewise contributes materially to the occurrence of colicky affections. As shown by the experiments of Ellenberger, Tangl and Scheunert, part of the gastric contents pass into the intestines very early, even towards the end of a meal; the food is chewed only once and not particularly well prepared, it may consequently easily exert a deleterious influence upon the intestines, either by undergoing an abnormal fermentation and irritating the intestinal mucosa or by easily becoming inspissated. Part of the ingested water also enters the small intestine immediately.

Very important are the methods of using horses and the amount and character of attendance they receive. No other domestic animal is so much exposed to errors in diet as the horse, especially in big cities where horses often receive spoiled food or improper food substitutes; often their meal time is not observed regularly; livery stable and dray horses must often work immediately after having been fed. The use of the horse as a work animal also brings about its frequent exposure to the inclemencies of the weather and to cold.

An important rôle must finally be attributed to thrombosis of the mesenteric arteries which occurs practically without exception only in the horse, but is very frequent in this animal. This affection accounts for a considerable percentage of the "colicky" diseases.

Symptoms of Colic. Horses frequently look around towards the posterior abdomen; they trip about restlessly, groan, paw and stamp with their front legs, and kick against the abdomen with their hind legs; if they become quiet for a time, they stand with the back arched, the neck and head stretched; they place the feet under the abdomen, switch the tail and are dull and apathetic. If the pains come on during motion, the animals have a short stiff gait; they do not like to move, soon stand still and can be made to move onward only by energetic urging.

During the periods of restlessness the animals try to lie down, doing so with great care; they may rest upon the abdomen with their legs drawn under it, or they rest upon the back or roll from side to side. In other cases patients suddenly and recklessly throw themselves down upon the ground, kick with the feet and roll very recklessly. Often the animals assume abnormal positions; they kneel on their front legs or sit on their haunches; they stretch out their front legs and elevate the thorax and head (sitting dog-fashion) or they stretch out their front legs and rest on the sternum.

The symptoms usually come on in the form of paroxysms, between which the animals are quiet and worn out; frequently,

however, they recover sufficiently and begin to eat, but a renewed attack soon interrupts the meal. Sometimes the restlessness is remarkably violent, the patients continually change their positions with hardly noticeable pauses; they constantly throw themselves on the floor and assume various positions; jump up again, kick, paw, rear and bite into various objects within their reach. During such violent attacks one may observe grating of the teeth, trembling, nervous movements (pressing forward, circulatory motions, shaking of the head).

Symptoms on the part of various organs depend upon the causative morbid conditions. These symptoms have been described in the preceding chapters.

Differential Diagnosis. Whenever symptoms of colic are present, it is always necessary to examine methodically all of the organs which may be the seat of the underlying affectiou, and to arrive at a differential diagnosis according to the principles laid down in the preceding chapters. Sometimes it may be necessary to ameliorate or remove a dangerous symptom even before the institution of the general examination.

Symptoms of restlessness may be caused by the following morbid conditions:

1. Diseases of the stomach: Acute and chronic gastric dilatation (overloading of the stomach, including stenosis of the pylorus), gastritis, gastric ulcer, parasites in the stomach.

2. Intestinal diseases: Acute enteritis (rheumatic intestinal spasm, spasmodic colic), occasionally chronic intestinal catarrh, enteritis (including specific acute infectious diseases and inflammations caused by poisons), meteorism, impaction, internal obstruction, stenosis, thrombosis of mesenteric vessels, strangulation (also incarcerated abdominal hernia), torsion and volvulus, invagination, intestinal worms.

3. Acute peritonitis.

4. Diseases of the liver and bile ducts: Lodgment of gallstones, acute, occasionally also chronic hepatitis, rapid increase in the size of the liver (for instance in consequence of hemorrhage), rupture of the liver.

5. Diseases of the urinary organs: Acute nephritis, nephritic abscess, pyelitis, obstruction of the ureter, inflammation of the bladder, occlusion of the urethra.

6. Diseases of the sexual organs: Torsion of the uterus, spasm of the uterus caused by movements of the fetus, labor pains in normal delivery and in abortion.

7. Diseases of other organs: Pleuritis (very rare!) certain diseases of the esophagus (spasm, obstruction, stenosis, dilatation), irritation of the rectum and its neighborhood by worms (oxyuris, gastrophilus larvæ), paralytic hemoglobinemia; also long-continued hunger and great exhaustion from work.

In making a diagnosis, symptoms of colic are only of miuor

importance, because they do not permit the determination of the underlying individual affection, or even of a group of allied affections. Almost continuous restlessness is, however, observed most frequently in acute gastric dilatation, in displacements of the intestines, in the grave forms of thrombosis of intestinal vessels and in certain forms of obturation of the intestines. Unnatural positions are seen, especially in forward dislocation of the diaphragm, in intense intestinal tension or pain in the abdomen (displacements of the intestines, impaction, intestinal calculi).

Icteric discoloration of the mucosa with simultaneous restlessness points to disease of the duodenum, of the bile ducts or of the liver.

Elevation of temperature from the onset points to an inflammatory or infectious character of the disease, while a subsequent rise of temperature may be caused by a secondary inflammatory process.

In cases which have not yet progressed too far, a nearly normal frequency and character of the pulse permits the exclusion of grave inflammatory and infectious processes, also pressure upon the diaphragm, and usually also displacements of the intestines and the grave types of thrombosis. In the further course of the disease and in young animals these features can, however, not be utilized for a differential diagnosis. The distribution of surface temperature usually runs parallel with the character of the pulse and with the condition of the respiration. The examination of the thorax occasionally furnishes data for a diagnosis of diaphragmatic hernia.

Belching, retching, vomiting and a sour smell of the expired air speak in favor of primary or secondary affections of the stomach.

Enlargement of the abnormal circumference may be absent in primary or secondary meteorism or it may be hardly observable if the abdominal walls are very tense or if the meteorism is confined to a small portion of the intestine; marked extension of the abdomen is usually caused only by bloating of the large intestine.

Percussion of the abdomen hardly assists in arriving at a diagnosis.

The intestinal sounds are of greater prognostic than diagnostic value. The absence of peristalsis in certain portions of the intestines cannot be determined from the behavior of the intestinal sounds, because, unless quite feeble, the latter are conducted from one spot to all parts of the abdominal wall. If, however, the sounds are persistently more frequent, and loudest over certain sections of the abdomen, one may conclude that the intestines of this region are in a condition of energetic peristalsis.

Complete constipation, coming on simultaneously with or soon after restlessness, speaks for obstruction or paralysis of

the intestine; the absence of these symptoms at the beginning does not, however, exclude these affections. Retarded defecation, and even complete constipation, is observed also during the later course of disease of the stomach. Marked straining at defecation is observed most commonly in diseases of the rectum and in peritonitis. The character of the feces assists in some cases in arriving at a diagnosis.

Rectal examination furnishes the most valuable data and it should never be neglected, as many cases can be diagnosticated correctly on the basis of a proper rectal examination.

To make a careful **rectal examination** one should first inject two or three quarts of lukewarm water into the rectum; this relaxes the rectal wall and makes its internal surface slippery. Before introducing the hand the neighborhood of the anus is inspected and tumors, parasites or blood are noted if present. The oiled or greased hand is introduced by overcoming the resistance of the rectum with the fingers closed into a cone. It is then advanced carefully until the arm has been pushed in as far as the elbow; while this is being done one can ascertain, by palpating the rectal wall, whether the latter has been torn. The hand then progresses in the direction of the thorax to find out whether the rectum is empty (obstruction). Then palpation is made through the rectal wall in order to examine the urinary bladder, the two abdominal rings (in mares the ovaries), the accessible portions of the small and large intestines, the posterior or mesenteric root, the posterior, upper portion of the spleen, the left kidney and, in horses that are not too large, the anterior mesenteric root and the vessels contained in it. A man of medium size is able, in not too large horses, after introducing the arm up to the shoulder, to palpate the anterior portion of the left kidney and from there downward and laterally a spherical segment of the abdominal cavity. If possible the examination is made on the standing horse. Very restless horses must be first quieted by an injection of morphine or by choral hydrate, or they must be restrained.

Treatment. In order to avoid serious errors, treatment must always be directed against the underlying disease, which has either been determined with certainty or at least with good probability. The variable nature of the causes of colicky pain excludes any uniform method of treatment; it is, however, always advisable to take the patients to a roomy closed place well littered with dry straw, to prevent reckless throwing down and impetuous rolling partly by narcotic means, partly by proper attendance, to prevent if possible ruptures and displacements (see chapters on internal strangulation, torsion and volvulus). An exception in this respect are torsion of the large intestine when moderate rolling may be permitted. The indiscriminate use of laxatives and similar drugs is absolutely worthless, frequently even harmful.

(Zschokke in general recommends sedative treatment in colics, particularly with tincture of opium (50 to 70 cc.) in camomile infusion, since in the course of 6 years he has observed a mortality of only 7.4% in cases of colic at the Zurich Clinic, in contrast with a mortality of 12% during the 5 previous years when he used the excitatory therapy with injections of arecolin and pilocarpin.)

Aside from the internal treatment of the underlying disease, operative interference will play an important rôle in the future; this includes the introduction of the stomach tube, lavage of the stomach, the reduction of displacements, the removal of obturating foreign bodies per rectum or by rolling, and finally laparotomy and enterotomy. One must, however, al-

ways remember that laparotomy, and still more laparotomy combined with enterotomy, are procedures that are quite dangerous for the horse and they must still be perfected more before many animals can be saved by them.

It is possible to diminish the colicky affections materially by a suitable prophylaxis, especially by dietetic measures (see page 406).

28. Animal Parasites in the Stomach.

(a) Gastrophilus Larvae in the Stomach of the Horse.

Occurrence. The larvæ of bot-flies occur almost exclusively in the stomach of horses after prolonged pasturing; they are, however, seen occasionally also in horses which have been working along roads along which much brush grows. Thus, of 437 horses examined at the abattoir in Augsburg, 142 (32.5%) were infested with Gastrophilus larvæ, as follows: 7.4% of the city horses, 17.2% of the army horses, 34.7% of the country horses and 37% of the horses from incorporated suburbs of the city (Stroh). In Nevada (in North America) 80% of the horses are infested with the larvæ of Gastrophilus nasalis and only 7% are not infested. Foals from eight months up to the age of three years are most commonly affected. Under special conditions, as in time of war, when the animals are constantly kept in the open, numerous cases may also occur in adult horses. Thus during the World War Larisch observed Gastrophilus larvæ in 60% of the German army horses on the Russian front. The number of cases increases particularly during dry years.

Etiology. Fly larvæ develop from the ova of brachyceric flies (Diptera brachycera) which include the bot-flies (Gastrus, Gastrophilus). The fullgrown larvæ are brown to red to yellowish gray, 15-20 mm. long, elongated, pointed in front, obtund behind, they are made up of 11 rings which are usually rounded more on the dorsal surface and beset with hooks. The first ring bears two chitinous mouth-hooks with wart-shaped antennae.

The larvæ of the following species of Gastrophilus occur in the stomach of the horse:

1. **Gastrophilus equi.** (G. intestinalis, large stomach bot-fly): it is 12-14 m. m. long, rusty-yellow, similar to a bee; it deposits its eggs preferably on the anterior part of the animals. The larvæ usually are attached to the cardial portion of the stomach, they are 18 m. m. long, thick, yellow-brown to flesh-colored, fitted with two rows of hooks which become larger anteriorly and are attached to the anterior margin of the body-segments; the 9th of these segments does not carry hooks in the middle of the dorsal surfaces, the 10th none on the back, and the 11th none at all. The fly is prevalent almost everywhere and gives origin to the largest number of stomach bot-fly larvæ.

2. **Gastrophilus haemorrhoidalis.** (Rectum bot-fly.) It is 9-11 m. m. long, very hairy, brown-black, also widely distributed. The female deposits her eggs preferably about the lips and nares at the basis of the hairs, but also on other places of the fore part of the body. The larvæ are only 13-16 m. m. long, dark red, without hooks in the middle of the dorsum of the 8th and 9th and frequently the 7th ring.

and without hooks on the entire dorsum of the 10th ring. The 11th ring is quite bare of hooks. The larvæ live in the cardial part of the stomach but also in the pyloric portion and in the duodenum; before being discharged, they remain in the rectum for a time, or even on the anus, and assume a greenish color.

3. **Gastrophilus pecorum.** (Cattle bot-fly.) It is 12-15 m. m. long, rusty-yellow to dark brown; the eggs are black. The larvæ are 13-20 m. m. long, dark red. There are no hooks on the back of the 6th and 7th ring in the middle, none at all

Fig. 30. Larvæ of Gastrophilus pecorum. Left dorsal, right ventral surface. Natural size.

on the back of the 8th-10th rings; on the lower surface of the 10th ring there is only one row; the 11th ring has no hooks at all. They live, in Hungarian and Russian horses, preferably in the stomach and duodenum, and also remain in the rectum for a time before being discharged (Fig. 30).

4. **Gastrophilus nasalis.** (G. veterinus, nose bot-fly.) This fly is 12-13 m. m. long, chestnut brown; the eggs are white. The larvæ are 13-15 m. m. long, whitish yellow, the posterior end reddish. They carry only one row of slender hooks, which are lacking on the back of the 9th ring in the middle. On the back of the 10th and on the 11th ring they are absent altogether. The larvæ live almost exclusively in the duodenum near the pylorus, only exceptionally in the stomach.

5. **Gastrophilus flavipes.** Occurs in the regions on the shores of the Mediterranean and in Asia Minor, the larvæ live in the stomach of asses.

6. **Gastrophilus inermis.** The larvæ were found by Brauer and Henry in the rectum of horses.

Fig. 31. Ovum with larvæ of Gastrophilus equi. Figures to the right double magnification, to the left highly magnified.

Evolution of the Gastrophilae. The eggs are elongated conical and are glued to the hairs in a downward direction (Fig. 31); as they open after 4 to 5 days, or according to Joly only after about three weeks, slender larvæ come out which consist of 13 segments. These bore with the anterior end into the mucous membrane of the place where they remain definitely, that is usually in the stomach; there they attach themselves by means of their hooks, and pass through a period of development of some 10 months. After being matured they detach themselves from the mucous membrane during the months of May and June, and enter the pupa stage in horse manure or in the ground, in the course of 1 to 3 days; after 30 to 40 days more they are fully matured flies and cohabit.

Evolution of the Gastrophilidæ. The elongated, conical eggs which are provided with a lid are deposited in the morula stage and are cemented to the hair in a downward direction (Fig. 32). Toward the third week the transparent embryo becomes visible and in the

third to the fifth week after oviposition it reaches complete development. In warmer regions the embryo seems to hatch sooner. Bedford observed hatching in South America between the seventh and the thirteenth day; Osborn observed hatching on the tenth day, though most frequently after the fourteenth day. After the larvæ (which are similar in appearance to ordinary fly larvæ, transparent, 1.3 mm. long) have reached their place of habitat in the stomach or intestine, they pass through two stages of development which are interrupted by 2 moltings. At the end of the first stage they reach a length of 6 to 9 mm., still resemble fly larvæ, are already light in color in front, the posterior one-half blood red in color and provided with heavy stigmal plates. When fully grown the larvæ are of a color and size characteristic for each species, are short and thick, barrel-shaped, cut off square behind and now have massive stigmal plates. After about 10 months the larvæ loosen their hold on the mucous membrane, usually in the time between May and September, particularly in May and June, pass out with the feces, particularly in the early morning hours, burrow into the ground, and pupate in 1 to 2 days. At the end of 30 to 40 days (according to Scheben and Schwah, in 6 to 8 weeks; according to Brauer, in 4 to 6 weeks; according to Hobmaier, in 32 to 56 days) the adult fly emerges and a few hours later copulates. Accordingly, the cycle of evolution lasts 1 year, of which 10 months are spent in the host.

There are frequent departures from the usual cycle of evolution. A few fully developed larvæ may leave the stomach of the host at unusual seasons, and after passing through the intestines they attach themselves to the mucous membrane of the rectum (November to April). On post mortem, larvæ in different stages of development are found side by side in the stomach all the year round (Hobmaier, Stroh), which is partly due to the difference in time when they are taken up in summer or autumn and partly to the difference in time required to complete evolution. Numan observed the passage of an already pupated larva.

During oviposition the fly, if she is not disturbed, hovers a few centimeters from the skin and with great regularity deposits 50 or more eggs on the hair in a place the size of the palm of the hand (each fly deposits from 400 to 500 eggs). If the fly is disturbed she will in her restless flight deposit her eggs scatteringly over the entire surface of the body of the horse and hover around the animal at a distance of one meter or more. The eggs are fastened to the hair by means of a glue which quickly dries in the air. The union between the egg and the hair is so firm that it is easier to pull the hair out by the root than to detach the egg from it.

The bot-fly larvæ are taken up from the month of June to the beginning of October, preferably during hot days of July, August and September (in South Africa from January to May, particularly from February to April), at which time the bot flies swarm with a buzzing sound (only Gastrophilus nasalis does not buzz), seeking horses, asses and mules that are kept in the open. In the eggs attached to the hair the embryos remain alive for weeks. Contrary to the original general assumption that the larva slips spontaneously from the eggshell, Roubaud and Hobmaier have shown that the larva emerges only when the lid of the eggshell is open either by the action of dampness or by pressure which frequently happens to strike the favorite places of attachment. This may come about by rubbing with the lips, gnawing, rubbing or biting one another, to which the horses are possibly induced by the irritation produced by the

lively movements of the embryo in the shell. After the lid of the egg has been loosened through pressure with the lips or teeth, the embryo rapidly emerges from the shell and immediately pierces into the epithelial layer of the mucous membrane of the lips or gums which come in contact with it. At other times it remains halfway protruding from the eggshell and finally falls to the ground (Hobmaier), where it remains alive for 2 days and, accordingly, may be taken up by the animals by sniffing on the ground or with the feed. In either case the larvæ burrow immediately under the epithelial layer of the mucosa, migrate below the epithelium toward the pharynx and at the end of the first or second day gain entrance into the stomach by the act of swallowing; some, however, may become attached in the pharynx as well as in the esophagus. During their migration in the mucosa of the mouth, they produce a troublesome itching.

Pathogenesis. Bot-fly larvæ may be present in the stomach, even in considerable numbers (according to Hobmaier's observation in 80 adult horses, a total of 30 to 200 in the stomach and duodenum), without disturbing its function markedly; this may be due to the fact that they are most commonly adherent to the left half of the stomach which plays no important rôle in the digestive function. If, however, they are present in very considerable numbers (according to Hobmaier, over 200), and when located, at least partly, in the glandular portion of the stomach they will cause disturbance of health, as will a smaller number located predominantly in the glandular portion of the stomach; furthermore, in young colts as also in poorly nourished animals (scarcity of feed, hard work, other diseases).

At each of the points of attachment, which lie about 1 cm. apart, a craterlike pit will be formed by the hooks of the mouth and by pressure of the head of the larva. In the neighborhood of the defects which they have produced in the gastric mucosa, the larvæ cause a chronic inflammatory process by poisonous metabolic products and by mechanical irritation. When the loss of substance is very considerable, the power of resistance of the gastric wall is very much decreased and an accumulation of gastric contents may easier occur. In exceptional cases the larvæ may perforate the wall of the stomach or even that of the duodenum (Numan), or some larger blood vessel is injured and hemorrhages may occur, which may have a fatal termination (Zürn, Dammann, Hertwig, Bruckmüller, Thomas). As to the pathogenic effect, the larvæ adhering to the pylorus and the duodenum are the most dangerous (G. hæmorrhoidalis and nasalis).

The ulcers caused by gastrophilus larvæ may, like any other loss of substance, form the portal of entrance for various infectious microorganisms (Perroncito).

When a massive invasion has taken place, the numerous larvæ which are always closely packed in the cardia portion of

the stomach, and still more those that are distributed over the entire mucous membrane of the glandular portion of the mucosa, interfere with the motor and secretory activity of the stomach to an equal degree, thereby causing digestive disturbances which are augmented by a gastric and duodenal catarrh which follows the invasion of the larvæ, or by a secondary bacterial invasion. The numerously attached larvæ may furthermore lead to a stenosis of the pylorus or duodenum.

Whether the hemolytic substance found in the larvæ by Weinberg, and more recently by K. R. and R. Seyderhelm and by de Kock, and which, according to the latter observers, is contained in the excretions of the larvæ, influences the character of the blood of the host after absorption by the gastrointestinal tract has not been definitely determined. Subcutaneous or intravenous injection of this substance (extracted from the body of the larvæ with water), which K. R. and R. Seyderhelm have called oestrin, produces a severe anemia similar to progressive pernicious anemia, which, in the horse, however, according to Klempin, later disappears.

Anatomical Changes. The bot-fly larvæ are found in the stomach in varying numbers. Their number may vary from a few specimens to 200 or 500 or 750 in fatal cases (Larisch). Numan counted 1,000 in one case, Velu more than 1,000 and Wright 1,030. In Kersten's case they filled 4½ quarts, while Rexilius once found the stomach and duodenum and Kater the duodenum filled entirely with Gastrophilus larvæ. Larisch often counted 250 to 350 larvæ in the duodenum. As already mentioned, the larvæ are usually present in large numbers and are closely packed in the cardia portion of the stomach, while in the glandular portion they are scattered here and there over the entire mucous membrane. The round, craterlike pits (erosions and ulcers of bot-fly larvæ) in the mucous membrane have a diameter of 3 to 4 mm., a reddened base and round, thickened edges formed by the thickened epithelial layers. After the discharge of the larvæ the pits produced by them remain visible for some time, giving the mucous membrane a docklike appearance with a peculiar arrangement. Later characteristic, concentric, cicatricial knots take the place of the pits. Around the point of attachment of the larvæ an intense reddening and swelling develops, sometimes with small hemorrhages. In the glandular portion of the stomach and in the duodenum, inflammatory changes with edematous swelling of the submucosa vary with the number of larvæ which attach themselves. Under certain circumstances a deposit is formed in the mucous membrane as the result of epithelial necrosis. Also signs of beginning total perforation of the wall of the stomach or of the duodenum may be present. (Schlegel found in a horse with a massive invasion upon and under the gastro-

splenic ligament more than 6 fully developed larvæ and one between the two layers of the serosa. Cameron found two larvæ on the serosa near the implantation of the esophagus, the serosa around the larvæ being inflamed.)

Larvæ in different stages of evolution are found all the year round. As early as August there may be found, according to Stroh, by the side of fully developed larvæ, some young, blood-red larvæ of the new invasion. By October the fully developed larvæ have entirely disappeared, while among the young remaining ones decided differences in size are found, and after the middle of November some are more or less fully developed. By February and March development is probably complete.

Symptoms. According to the intensity and the time of the infection and the nutritive condition of the affected animals, the symptoms come on either towards the end of the period of pasturing or shortly after the stabling of the animals, or occasionally only during winter. In the foreground of the clinical picture are variable appetite, poor appearance of the animal, pale discoloration of the mucosæ, marked emaciation, debility and a peculiar luster of the eyes, toward the end decided weakness and attacks of colic, which, however, according to Larisch, are observed only when Gastrophilus pecorum and G. nasalis are present. The heartbeat becomes thumping, the pulse weak, the general condition poorer and poorer, and the animals, which finally suffer from a complete lack of appetite, have dwindled down to mere skeletons, so that they finally cannot get up from the floor any longer. If proper treatment is not instituted the animals may die from six to eight weeks after an intense invasion (Kröning) or perhaps only after two to four months; in milder cases spontaneous recovery occurs. Kater observed in a horse the picture of stenosis of the duodenum; Tegg that of pylorus stenosis; Rexilius saw increasing cachexia, lack of appetite and increased thirst.

The presence of larvæ of Gastrophilus hæmorrhidalis and G. nasalis causes an irritable condition of the rectum, which leads to symptoms of gastric catarrh (frequent defecation, straining, itching, eventually excitement and restlessness, similar to those in colic); straining may exceptionally lead to prolapse of the rectum.

In a case reported by Jacobsen, a Gastrophilus larva had attached itself behind the prepuce, thus inhibiting copulation, since every erection caused the animal pain. A similar result was observed in a case reported by Aichmayer, in which the larva had burrowed into the penis behind the glans. In both cases the disturbance was relieved after the larva had been removed.

Gillet observed the evacuation of fly larvæ with the feces (myiasis intestinalis) which had hatched in the intestinal tract from eggs deposited on moist bran by the house fly. No disturbance of health was observed.

Diagnosis. The greatest significance must be attached to the history of the case (onset of the disease shortly after a rather long period of pasturing) with a negative result of the examination of the various organs. Additional evidence is forthcoming if Gastrophilus larvæ are expelled from time to

time per rectum or if larvæ are found attached to the mucous membrane of the rectum (in the region of the so-called "Mastdarmrose"). In all suspicious cases a rectal examination should be made, since in cases of massive invasion bot-fly larvæ in different stages of evolution will also be found attached to the mucous membrane of the rectum at any season of the year but especially in fall and winter. The practicability of the allergic reaction successfully used by Van Es and Schalk, by the instillation of a few drops of a watery extract of the larvæ into the conjunctival sac, must await further tests, the more so since Luehrs could not corroborate the practicability of the test. (The larvæ of Gastrophilus equi and G. nasalis were each ground up separately, treated with 40 cc. saline solution, both extracts united, and the mixture filtered through cotton and heated at 60° C. for one hour.) Cachexia of foals can be excluded by the fact that it disappears entirely during pasturing or at least improves considerably. The presence of ascaris in the intestines can be ascertained by finding the worms of their ova in the feces. The exclusion of sclerostomiasis is always very difficult and often can be made only on postmortem examination; both diseases may occur simultaneously. Infectious anemia attacks horses without difference of age and, in chronic cases which alone have to be considered with reference to differential diagnosis from gastrophilus infection, causes repeated attacks of fever with little disturbance of appetite.

Treatment. Certain removal of all bot-fly larvæ from the stomach and intestinal tract can be accomplished by the administration of carbon bisulphide (carboneum sulfuratum, spiritus sulfuris), as first recommended by Perroncito and Bosso, later by Larisch and by K. R. and R. Seyderhelm, but especially improved by Hall, Smead and Wolf and Hall and Wigdor. According to the experiments of the American observers, which were checked by post-mortem examinatiou, the best and the least drastic dose for adult horses and colts over 2 years old is 21.5 cc. (28 gm.) in hard gelatin capsules. However, one may also give 2 doses of 14.5 cc. (18.5 gm.) each with an interval of 2 hours, or 3 doses of 10.8 cc. (14 gm.) administered at intervals of 1 hour. Colts under 2 years old should receive one-half or one-third of this dose. All feed is withheld from the animal beginning at noon, and on the morning following the drug is administered on the empty stomach, whereupon the animal is fasted for 4 hours longer. It is not necessary to administer a laxative (250 to 500 gm. castor oil, 6 to 10 gm. tartar emetic in sweet milk or some mucilagenous substance, 15 to 30 gm. aloes in pills, or up to 1,000 grams linseed oil). The administration of oils within the first half hour after the administration of the carbon bisulphide even has a decidedly deleterious effect upon the curative results. Such drugs should

not be administered until 4 hours later. The evacuation of dead larvæ, when no laxative is administered, begins on the following day, reaches its height on the third and fourth days, and scattered larvæ may even appear from the tenth to the fourteenth days. The expulsion of larvæ will be accelerated by the administration of laxatives (beginning within 24 hours and terminating in 5 days). Young larvæ are more sensitive to the drug than adult larvæ. For heavy horses Larisch recommends 3 capsules of 10 gm. each at hourly intervals, for light horses 2 capsules of 10 gm. each and 1 of 8 gm., and for very small horses 2 capsules of 8 gm. each. A very mild method of administration, which is especially recommended for weak animals, was reported by K. R. and R. Seyderhelm: Feed and water are withheld (water is allowed in the morning); on the following morning the capsules are administered, and 4 hours later 3 liters of wheat bran and 3 liters of warm water are given to the patient. Next morning an aloe bolus is given on the empty stomach; at noon and night 4 liters of wheat bran with 2 liters of lukewarm water are allowed, after which the patient is watered. On the second day following give 3 times daily 4 liters of bran moistened with water, hay and drinking water.

The medicine is administered with the aid of a balling gun or the capsules may be lubricated with castor oil or vaselin, and placed on the tongue by the hand, provided the animals are not too young; they are then swallowed by deglutition due to reflex irritation. One may improvise a simple balling gun by splitting crosswise one end of a reed 12-15 cm. long; the four sections made by the cross incisions must be rounded off. One end of the capsule, made slippery as indicated above, is grasped between the four sections of the reed and is pushed along the hard palate, back to the velum, then the capsule is swallowed (Tarr). On the day of treatment and on the following day the appetite is depressed and about 30% of the animals will refuse two meals entirely. The treated animals should be rested for 3 days.

Animals which are greatly emaciated are much affected by energetic treatment and some may even succumb to it (authors' observation). Santy found oil of turpentine effective (50-80 gm.); it is given in milk. Jacobsen uses coal tar (15-20-30 gm.) with linseed gruel three times during one day, the last dose being combined with 5-10 gm. of aloes. Allesandrini recommends chloroform to be given in the same manner as the carbon disulphide (not effective, according to Hall).

Larvæ in the rectum may be removed manually or by injections of soap-, vinegar-, or creolin-water; also with carbon disulphide (5 to 10 gm. to one quart of mucilage). Labat recommends the inunction of the rectal mucosa with boro-vaselin, whereupon the larvæ become detached, without dying, and are voided. The discharged larvæ should be destroyed.

Prophylaxis. This consists in the destruction of the evacuated and artificially removed larvæ, careful grooming, especially from June to the beginning of October, and whenever possible postponement of grooming from the early morning hours of the day to late in the evening (early opening of the eggs before the embryo is fully developed). Whenever possible the eggs should be removed daily from the hair. In the temperate zones this should be done at least every 8 days. The hair on which eggs have been deposited may be cut with scissors or the eggs scraped off with a knife and burned. This should be done outside of the barn, at the feeding places, hitching places, and at pasture. It is well at the same time to rub the more distant parts of the body (fetlock, coronet) with a rag soaked in kerosene in order to destroy the larvæ that have already hatched. In case no suitable place to groom the animals is at hand, they may be placed on an old wagon sheet, which, after grooming has been completed, should be carefully rolled up and disinfected at a proper place. It seems well to undertake in October the treatment of all horses that have had an opportunity to ingest bot-fly larvæ.

Nematodes in the Stomach of the Horse. The finely filiform **Spiroptera megastoma** causes the formation of firm worm-swellings, in size up to that of a hen's egg, rarely larger, which are constructed in separate compartments; they also lead to adenoma-like swelling of the glands in the mucous membrane. The swellings are almost without exception situated beneath the mucosa which contains the glands, near the folded border, and are covered by a normally tinged mucous membrane with one or several openings from which purulent secretion and worms may be pressed out. The worm-swellings make their appearance particularly in the month of July, and are very frequent in New South Wales; they seem to cause digestive disturbances only rarely, if they are very large and if situated in the region of the pylorus (Railliet, Knabe, Fiebiger); but the origin of the digestive troubles can hardly be ascertained during the life of the animals, because the embryos, which are 0.6-0.7 mm. long, leave the eggs in the mother animal, and it is very difficult to discover them in the fecal masses. For the diagnosis of hebronemiasis Hodgkins recommends allowing only gruels for two days followed by a fast for 36 hours. The stomach is then irrigated with 15 liters of lukewarm water. The worms will be found in the last remnant of water siphoned off. According to Berké individual Spiropteræ megastoma may move about freely in the stomach and intestine and attach themselves to the mucosa; according to Weston they may produce ulceration in the pyloric region of the stomach and in the gut, followed by peritonitis.

The **Spiroptera Microstoma** appears to be more harmful. It sometimes lives in large colonies of individuals free in the contents of the stomach, often has been found imbedded deeply in the mucous membrane by means of its anterior end, and in this case it leads to ulceration (Railliet) or thickening of the mucosa (Dupuy and Prince). Diem observed in four horses slight symptoms of colic during eating, also vomiting, emaciation and general weakness; these symptoms disappeared after the spiroptera, which were not determined particularly, had been removed by drugs. Hodgkins repeatedly saw cases of gastritis verrucosa with marasmus after the immigration of numerous Hebronema (H. megastoma, H.

microstoma, H. muscae). Diem found the carbon bisulphide and aloes treatment as used for gastrophiliasis very effective in treating spiropteriasis. Ransom and Hall also recommend oleum chenopodii anthelminthici. According to Martin's experience the stomach spiropterae are very resistant to the usual anthelminthics, but large doses of lysol are said to cause their discharge.

According to the investigations of Ransom and Hall, the evolution of the stomach Spiroptera is as follows: The embryos of Hebronema megastoma and H. muscæ which have been evacuated in the feces burrow into the eggs and larvæ of the house fly, those of H. microstoma into those of the stable fly, and continue their development in these. In the adult fly they are found especially in the head, but also in the thorax and abdomen. The flies are swallowed by horses with the feed and water, and the larvæ thus reach the stomach of the horse, where they continue their development to sexually mature worms. According to Descazeaux, strayed Spiroptera larvæ cause the so-called summer sores or dermatitis verminosa.

The **Trichostrongylus Axei** (Strong. Axei. Str. tenuissimus) has been found in adenoma-like formations in the mucous membrane of the stomach (Axe, Petit and Germain, Wolffhuegel, Mazzanti).

Seurat found Spiroptera sexalata (Physocephalus sexalatus) with the front end embedded in the mucous membrane of the stomach of the ass and the abomasum of the dromedary. The embryo is said to leave the egg in the intestinal tract of an insect.

(b) Stomach-worm Disease of Sheep and Goats. Strongylosis ventriculi ovium et caprarum.

Occurrence. The disease occurs in marshy territories, exposed to frequent rains and inundations, and it sometimes causes great losses. Animals of all ages are affected, but preferably only lambs, kids and yearlings. According to Guberlet, the frequency of infestation with stomach worms may change with the seasons, as in some regions they make their appearance in spring or early summer after the animals have been placed upon infested pastures and disappear again in fall and winter when the animals are on dry feed, while in stabled animals the disease is seldom observed. The affection is frequently seen simultaneously with the lung-worm-, or the liver-fluke disease.

Fig. 32. Anterior extremity of Haemonchus contortus with teeth-like papillæ arranged in a posterior direction.

Etiology. The disease is produced by stomach strongylides which belong to the subfamily of the Trichostrongylides. Of primary importance is Strongylus (Haemonchus) contortus, for which reason the disease is also known as haemonchosis, but other stomach strongylides are usually of etiological import together with it.

The **Strongylus (Hæmonchus) contortus** Rud. is a filiform whitish or red worm, the red color of which is attributed by most authors to blood coloring matter which it has sucked up, although Lignières among others believes it to be a different coloring substance. Near to the frontal end, the worm carries two lateral tooth-like papillae; the mouth is bare. The male is 1-2 cm. long, its bursa consists of two long lobes and an accessory right lobe. The female is 2-3 cm. long, pointed at

the posterior extremity; the vulva is situated in the caudal fifth of the body, with a wart on each side. The eggs are elongated oval; at the time of deposition they are grooved, according to Railliet they already contain an embryo.

Strongylus (Nematodirus) filicollis Rud. is a hair-shaped white worm, very thin in front, thicker behind, with longitudinal ridges and bare mouth. The male is 0.8-1.3 cm. long, the bursa two-lobed with two very long spicula. The female is 1-2.4 cm. long; the vulva is behind the middle of the body. The eggs (Fig. 35) are elongated oval, 110-113 μ long, grooved.

Of rarer occurrence are Strongylus (Trichostrongylus) retortaeformis, Str. (Trichstr.) instabilis, Str. (Ostertagia) Ostertagi, (Str. convolutus), Str. circumcinctus s. vicarius (Ostertagia circumcincta), Str. (Cooperia) Curticei (Str. ventricosus), Str. subtilis (grecilis).

Evolution of the Strongylides of the Stomach. The eggs passing with the feces (a moderately infested sheep passes 3 million eggs daily) are in the morula stage (Fig. 33-35) or already contain embryos. Within a few hours to one day and at a proper temperature (5° to 10° C.) an embryo 0.3 to 0.4 mm. long is formed, which soon leaves the eggshell. They will attain a length of 1 mm. at an outside temperature of 39° C. in 4 days, at 21° in 6 to 14 days, and at 10° in 3 to 4 weeks. In the meantime they pass through two moltings and thus become capable of infesting another animal. After reaching the abomasum or small intestines of a suitable host they become sexually mature in 2 to 4 weeks. According to Boulenger, Strongylus filicollis forms an exception to this cycle of evolution, in that this strongyle passes through one molting in the eggshell when still in the embryonic stage, and in this encysted condition is able to resist drying out and high temperature for 20 months. Under favorable conditions, however, it leaves the eggshell in 24 to 28 days after oviposition. Accordingly in this strongyle not only the already hatched larvæ, but also those still within the eggshell will become sexually mature when taken up by a suitable host.

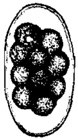


Fig. 33. Egg of Haemonchus contortus.

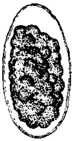


Fig. 34. Egg of Strongylus ostertagi.

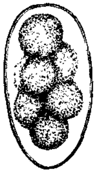


Fig. 35. Egg of Nematodirus filicollis.

According to the experimental observations of Lourens, the larvæ of Strongylus contortus, like those of the lungworms, can develop in about 4 to 6 weeks into a nonparasitic generation which multiplies by sexual copulation. The worms of this nonparasitic generation are only a few millimeters long, and by the continued deposition of eggs that are about one-third smaller than the eggs of the parasitic generation they increase greatly in number in the outside world. If soil be sterilized (autoclaved at 2½ atmospheric pressures at 134° C.) and grass allowed to grow in it and then inoculated with cultures of Strongylus contortus and the grass fed to lambs after the nonparasitic generation has developed, the lambs will become infested with stomach worms.

The resistance of the larvæ is very great. According to Ransom, they can stand desiccation for thirty-five days, and, according to Piana, up to ten months. They become immobile in much water; can, however, revive later on. They can stand the cold of winter without any harm and remain alive on pastures for seven or eight months. (Guberlet found live larvæ somewhat degenerated in cultures made in soil and feces after 16 months.) Embryos that are still in the

shell soon die when subjected to drying out or to freezing temperatures. At a temperature below 5° C. they do not hatch at all. The eggs of Strongylus filicollis remain capable of further development for 20 months and longer. The resistance of the nonparasitic generation is probably similar to that of lungworms.

Natural **infestation** takes place predominantly on pasture by the ingestion of encapsulated larvæ from eggs that have recently been passed with the feces of infested animals or by the ingestion of larvæ of the nonparasitic generation. The larvæ are taken up with either the feed or the drinking water. The infestation on pastures is favored by the fact that the larvæ will follow the moisture (dew, drops of water) that collects on the end of blades of grass at night and on rainy or foggy days. The pastures become infected year after year by sick animals or by apparently healthy infested animals (carriers), but also retain the infestivity from one year to the next by reason of the great resistance of the larvæ.

Infestation may also occur in the stable, as shown by the observations of Michalik and the experiments by Lourens, and is possible by eating from the ground, by ingestion of bedding and by drinking water from pools and ponds.

Pathogenesis. According to the general conception of the immigration of the larvæ into the abomasum, the larvæ locate directly in the abomasum or partly also in the small intestines. The experiments of Ransom on guinea pigs (presence of the fed Haemonchus larvæ in the lungs 48 hours after infestation), however, admit the possibility that the larvæ become permanently located in the stomach in a roundabout way by way of the lungs, like many strongyles of the intestinal tract. The strongylidæ bore into the gastric mucosa and suck blood from it; in this manner they disturb the nutrition of the hosts in a degree proportionate to their number. More detrimental than the loss of blood is, however, probably the absorption of toxic metabolic products (according to Grosso hemolysins) of the parasites.

The injuries produced by the parasites afford an opportunity for bacterial invasion. Lignières showed that a disease prevalent in Argentine and known under the name of Lombriz (see Vol. I) was caused by a bacillus bipolaris entering through such injuries made by Strongylus (Lignières).

Anatomical Changes. The parasites usually adhere in large numbers to the mucosa of the abomasum which presents the appearance of an acute or chronic catarrh, and occasionally also small ulcers. The contents of the abomasum may show a reddish discoloration. In a case reported by Kohn the mucous membrane of a grown goat appeared blood red with a brownish tone as a result of a continuous layer of similarly colored Haemonchus contortus. The presence of the stomach strongyles can sometimes be detected only by a microscopic examination of

the stomach contents (Stoedter), of scrapings of the mucous membrane or of a piece of mucous membrane squeezed between two slides (Wester).

Symptoms. In case of a massive invasion symptoms become manifest in 14 to 19 days after infestation. According to the time of ingestion of the larvæ, they are observed in the spring and early part of summer and also in late summer, fall or winter. The symptoms consist of disturbances in nutrition, debility, depression of appetite, bloating and severe diarrhea, which, in cases of a protracted conrse, has a tendency to improve periodically. There is also observed anemia, hydremia and finally cachexia: sometimes colic, convulsions or a paralysis-like weakness, and regularly catarrhal conjunctivitis with a discharge from the eyes. The blood shows many poikilocytes (deformed red blood corpuscles, Wernicke) on microscopic examination and basophilic granules in the red blood corpuscles. Kohn observed in a goat a strong desire to push forward, convulsions of the forelegs, lateral deflection of the neck and great emaciation.

The mortality amounts to 20% to 100% (Lourens), or again to 80% to 90% (Keller). Very young and weak animals will die at the end of one or several days as a result of acute diarrhea or loss of strength, while otherwise death does not ensue until after weeks or several months. In spite of their recovery and healthy appearance, the animals will remain carriers for 2 to 4 years (Lourens). In such animals Keller often saw continued emaciation with swellings of the head which finally terminated fatally.

Diagnosis. Diagnosis is confirmed by the cultural detection of the larvæ or by the microscopic detection of the eggs in the feces; also by post-mortem examination of animals that have died or have been slaughtered for that purpose.

For the cultural detection of larvæ as recommended in South Africa, freshly passed feces or feces removed directly from the rectum with a suitable instrument or simply with the finger are placed in a fruit jar. The lid is then replaced and the jar set aside in a dark place and observed day by day. If sufficient moisture is present (a fine deposit of moisture should form on the inside of the wall of the jar) the larvæ will be seen crawling up the glass wall beginning on the fourth day in the form of fine, whitish, branched, mucuslike streaks. When exposed to light they will crawl back into the fecal masses. In this manner the degree of infestation can also be determined.

For the microscopic detection of stomach-worm eggs and for an approximate determination of the degree of infestation the "agitation" method (see page 340) is practical. In case it is desirable to detect parasite eggs when only a small number are present, or if they are to be secured free from foreign admixtures, the enrichment method is used. With the method of Bass, recommended by Lewis, the feces are made into a thin paste and pressed through a piece of muslin to remove coarser food particles. The material is then several times shaken with large quantities of water, and after the mixture has settled the water is poured off until it finally remains colorless. The sediment is now taken up in aqueous calcium chloride solution of a specific gravity of 1.25, shaken up and centrifuged. The solid particles will sink to the bottom, while the lighter eggs of the parasites will collect on the surface of the specifically heavier calcium chloride solution, from where they may be removed with pipette. The

eggs are pipetted into a centrifuge tube and water is added to them. Upon centrifuging, the eggs will collect at the bottom of the now specifically lighter water without the admixture of fecal particles and can now be placed upon the slide for microscopic examination. According to the method of Guberlet, specimens taken from different places of freshly passed feces in the amount of 1 gm. each are weighed off, mixed with water, and passed through a series of sieves having 6 to 100 meshes to the inch. The eggs are not counted in each specimen. In drawing conclusions as to the degree of infestation, it should be remembered that there are 1½ to 2 times as many female strongyles as males, and that the proportion of the number of eggs counted to sexually mature worms is influenced by obstipation, diarrhea and starvation (compare also page 340).

Treatment. By the systematic and accurate experiments carried out by Veglia and Green and the practical results obtained in South Africa, the complete reliability of a uniform mixture (wire-worm remedy) of copper sulphate and sodium arsenate in the proportion of 4 to 1 in effecting a cure was shown, if the mixture was administered in 2 doses at 24 hours' interval. The dose is 0.625 gm. for adult sheep, 0.5 gm. for yearlings (2-tooth), 0.375 gm. for lambs 6 to 10 months old, 0.25 gm. for lambs 4 to 6 months old and 0.2 gm. for lambs 2 to 4 months old, given on the empty stomach. In South Africa the customary method is to administer the mixture in powder form. The mixture is placed in a container and each dose measured out with a special graduated spoon (in South Africa a set of 5 such graduated spoons is furnished with the mixture) holding the required dose when level full (levelled off by means of a straight edge fastened across the top of the container). From these spoons the mixture is emptied into an ordinary teaspoon and then placed as far back on the tongue as possible. The animal should be dosed in a standing position (see treatment of liver fluke disease) to prevent probable aspiration of the mixture. In order to prevent the inadvertent administration of two doses on the same day, each treated animal should be separated from the still untreated ones as soon as the dose has been administered. According to the author's own observation on successfully treated sheep, a more accurate dose can be administered more conveniently by dissolving the mixture in acidulated water. For this purpose the required dose is dissolved in 10 cc. of rain water regardless of the size of the animal. In the total amount of water thus calculated the required amount of the mixture for animals of the same size to be treated is dissolved, after the addition of 0.4% concentrated hydrochloric acid (about 14 cc. per liter of water), and 10 cc. of this solution is injected into the mouth of the animal with a Pravaz or Record syringe. The mouth of the animal should be held slightly upward and the solution injected at the commissure of the lips between the wall of the cheek and the molar teeth. It is practical for this purpose to attach a blunt hypodermic needle to the syringe and if possible to use a larger syringe (wound syringe) with 10 cc. graduation and provided with a stop-nut. (For treating large number of sheep with a

solution an inverted graduated glass cylinder is used in America. The neck of the graduated cylinder is closed with a triple-perforated stopper. Through one of the perforations is passed a glass tube connecting with the larger vessel holding the bulk of the solution, through a second perforation a tube attaching to the drenching tube with a metal mouthpiece, and through the third glass tube reaching to the bottom of the graduated cylinder for the admission of air.) In order to prevent the poisoning of a number of sensitive animals, only 10 animals should be dosed at first, and in case of a favorable outcome of this preliminary trial another 30 animals may be treated. If the second trial also results favorably, the rest of the flock may be treated. In case of severe cold or cold wet weather the animals should be kept in a barn or under some shelter for several days following treatment. If the animals are to be placed on infested pastures the treatments should be repeated at intervals of one month.

In detail the treatment of suckling lambs, which lasts 2½ days, is as follows: Separate the lambs from their mothers at 6 a. m. Four hours later give the first dose. Starve them four hours longer and then turn them in with their mothers till night. Separate the lambs again from their mothers during the night and administer the second dose the following morning. Starve the lambs for four hours longer and then again turn them in with their mothers. Sucking lambs should be treated a week later than their mothers.

The method of treating other sheep is as follows: Withdraw feed and water totally at 1 to 2 p. m. on the first day, and administer the first dose between 6 and 10 a. m. on the second day. Feed but do not water at 3 to 5 p. m. At 6 to 10 a. m. on the third day the second dose is administered, and the animal is fed but not watered at 3 to 5 p. m. Following this feeding, feed and water should again be withheld until the morning of the fourth day, after which the animals may be fed and watered in the usual manner. (The same concentration of the solution may also be given to groups of animals of different sizes, but in this case the size of the dose should be adjusted to the size of the animal.)

Another treatment which, according to Guberlet, has an efficiency of 90% to 100% in America, consists of a 1% solution of copper sulphate added to a 1% tobacco solution. Of this decoction lambs under 1 year receive 50 c.c. and sheep over 1 year 100 c.c. in one dose after they have been starved for 24 hours. According to Hall and Foster, a 1% solution of copper sulphate given alone is also effective. Von Linden recommends copper bichloride as a lick or 5 c.c of a 1% solution (see also treatment of lung worm disease). According to Hall and Foster, oleum chenopodii anthelminthici (0.2 c.c. per 1 kg. body weight in

emulsion with 150 c.c. milk) is also rather effective. The following drugs have also been used with more or less success: Oil of turpentine (5 to 10 gm.) with about 60 c.c. linseed gruel or together with rancid animal oil (āā by the coffee spoonful) 3 times in succession and with the last dose 0.3 to 0.5 gm. tartar emetic; kamala (4-5.0 gm. per day in water or milk), picrate of potash (0.12-0.20 gm. in an infusion of linseed on three consecutive days, Raabe), areca nut and also arsenic (for ten sheep daily 50.0 gm. of areca nut and 1-2 gm. arsenic mixed with bran, Moussu). Creosote (of a 1% aqueous solution, lambs 60 to 120 gm., adult sheep 90 to 150 gm.), which, according to Moussu, may also be offered the animals in ½% solution as a drink for periods of 3 days with equally long intervals (very sick animals will drink the solution voluntarily). Creosote may also be given in 1 gm. doses in gelatin capsules, or teaspoon doses of 20% creosote vasoliment, also 1.5 gm. creosote, 5 gm. extr. filicis and 3 gm. thymol with a little alcohol, gum arabic and 200 gm. of water. According to Craig, thymol alone also has a favorable effect when given to lambs in 2 gm. doses and yearling sheep in 3 gm. doses made into a paste with a little mucilaginous water and diluted with ¼ liter of water. Lysol (250 c.c. of an aqueous solution) has also been used successfully. All liquid vermifuges may also be administered by means of a rubber tube to one end of which a funnel has been attached, while the other end is introduced directly into the stomach, thus greatly increasing their effectiveness.

Prophylaxis. This consists in avoiding wet, marshy and suspicious pastures and the separation of the sick and of all adult animals from the lambs and kids. The animals should have access to good and, if possible, to running water; preferably there should be elevated stone steps leading to the source of water to prevent the animals from tramping into it or contaminating it with their feces (Stödter). If a continuous avoidance of infected pastures is not possible, they should be abandoned as such for one or two years or utilized for other purposes. Ransom recommends a procedure in this respect which promises permanent results, because the worm-larvæ die within one year if the infected pasture is not visited, in the meanwhile, by sheep or by susceptible animals. According to Lourens, this method has not proved successful, for many observations show that pastures that have become infested remain dangerous for many years. According to this observer, sheep and goats are best kept off such pastures permanently and all brought-in animals examined for stomach worms, and if found to be carriers isolated in a barn.

From October to March the animals may be kept in a common barn without consideration as to age, and they may be pastured together in April in a non-infected pasture. In May the pasture is changed every two weeks, and in June every tenth day, and between July and August every week in such a manner that

the preceding pastures are no more visited during the same year; in September the change of pastures should again occur in longer intervals. Next year the same pastures may again be visited in the same order of rotation, because the disseminated ova or embryos have perished in the mean time. In the fall the adult sheep should receive an anthelmintic course of treatment.

Another method consists in dividing the pasture by a small neutral strip into two portions, one for the lambs and one for the adult animals. The lambs should be allowed to be with their mothers only during sucking. The new-born lambs should be separated immediately from affected and suspected ewes, and should receive only boiled milk.

Stomach-worm Disease in Deer. The Strongylus contortus, Str. Ostertagi and the Str. filicollis sometimes produce stomach-worm disease in deer and occasionally leads to many cases of death. The affection gives rise to a hydremic cachexia if the invasion of the worms is massive. According to Freund, the stomach-worm disease is also observed in the camel and the dromedary.

(c) Stomach-worm Disease of Cattle. Strongylosis ventriculi bovum.

Occurrence. Strongylidæ are frequently parasitic in the abomasum of cattle, but they produce disease only if present in large numbers. The disease is then usually seen in young cattle after they have been pastured. Sometimes the disease occurs in an enzootic form (Harker, Penberthy, MacFadyean, Liénaux, Klein).

In the Berlin abattoir, Ostertag found Strongylus convolutus (Str. Ostertagi, Stiles) in 90% of the cattle killed.

Schnyder, who has studied stomach strongylosis, claims that the disease known in the country around the Zürich Lake as "*Kaltbrändigkeit*" is a strongylosis found during all seasons and in cattle of all ages, independently of dry or green feeding. He could demonstrate the presence of the disease in 0.2% of all cattle. Bang, however, calls attention to the fact that these cases, and others frequently observed in cattle, may be a combination of gastric strongylosis with the much more dangerous enteritis paratuberculosa Bang (see Vol. I).

Etiology. According to Schnyder's investigations there occur in the abomasum of cattle Strongylus (Ostertagia) ostertagi (Str. convolutus), Str. (Trichostrongylus) restortæformis, Str. (Cooperia) Curticei (Str. ventricosus), Str. oncophorus (Cooperia oncophora), and the Str. (Haemonchus) contortus Rudolphi. One usually finds several species simultaneously in the stomach and intestines.

Concerning the embryology of the gastric Strongylus of cattle, the mode of natural infection and the pathogenesis, the reader is referred to what has been said under strongylosis of sheep (see page 324).

Anatomical Changes. According to Schnyder and Blunschy, one finds in an advanced stage of the disease emaciation, anemia and eventually hydremia; the mucosa of the abomasum is swollen and edematous, raised in gelatinous folds, reddened here and there and beset with grayish white nodules, the size of a pinhead, with a small central pale vesicle (these formations are the so-called worm nodules). One also sees erosions from the size of

a lentil to that of a finger nail. Sometimes the mucosa is covered with a heavy flocculent deposit. The Strongylus worms may be found in the washings of the contents of the abomasum (decanting the contents).

The mucosa of the small intestines, especially in the median portions of the latter, here and there is swollen and loose, forms coarse transverse and longitudinal folds; it is reddened in spots as it is in enteritis paratuberculosa.

Symptoms. The clinical symptoms of gastric strongylosis of cattle come on late in summer or early in fall and are similar to those of the worm disease in sheep (see page 326). After mixing the feces with water and pipetting off the latter, one will find in it more or less numerous strongylus ova. During an outbreak of the disease in a pasture Christiansen saw 18 out of 67 calves die as a result of cachexia.

Watery stools are the most prominent symptom of the disease called *"Kaltbrändigkeit,"* the fetid stools are voided in a curved stream. Thirst is increased. The further course brings about rapidly increasing emaciation, anemia, diminution of the secretion of milk. There may be an edema of the external region of the larynx and of the thorax even during the first week of the affection. Temporary improvement occurs occasionally.

Diagnosis. A reliable diagnosis can only be made by excluding disease of all other organs and finding the ova in the feces; sometimes the correct diagnosis is made only after the animals have been killed or have died a natural death. The differential diagnosis has to consider particularly enteritis paratuberculosa (see Vol. 1).

Treatment and Prophylaxis. In cases not very much advanced Schnyder had good results from the administration of flores cinæ (150-200 gm. prepared as a decoction). One may also use the drugs mentioned under worm disease of sheep; the doses have to be increased proportionately (see page 327). Klein obtained favorable results with creosote.

According to Craig, good results also follow the use of thymol (yearling cattle 7.5 gm.), lysol (14 gm. in 1½ liters of water), copper sulphate (2 to 4 gm.), and arsenic (0.3 to 0.6 gm. with 7.5 gm. iron sulphate). Moussu recommends a mixture of acidum arsenicosum 0.5 gm. and 5 to 10 gm. tartarus stibiatus administered once daily mixed with bran, oats or turnips. If the animals do not eat well they should be dosed separately.

The prophylaxis calls for measures like those recommended against strongylosis of sheep (see page 329).

Amphistomum in the Stomach of Ruminants. Amphistomum cervi (conicum) is found in the rumen of cattle, sheep and goats. Its eggs are oval, 155 to 162 microns long, and provided with a lid at the smaller pole. Gastrophylax crumenifer is found in the rumen of the bison, cattle and zebu, and also in the

reticulum of the bison: G. elongatus, G. cobboldi, G. gregarius and G. minutus in the rumen of bison, zebu and gayal. Paramphistomum orthocoelium is found in the rumen of the bison, P. bathycotyle in the abomasum of the bison. Homalogaster paloniæ is found in the abomasum of the bison. Amphistoma seem to be harmless inhabitants of the stomach. Only in massive invasions of the zebu in Tonkin by Gastrophylax crumenifer was emaciation observed.

(d) Parasites in the Stomach of Swine.

1. **Spiroptera strongylina;** a slender whitish worm, males 10 to 13 mm., females 12 to 20 mm. long; it forms small tumors in the submucosa; it bores into the mucosa and may in this manner cause serious gastritis. This parasite has repeatedly caused enzootics, terminating fatally within three to four days (v. Ratz). Ciurea on the other hand, found in 9 out of 72 pigs 1 to 27 worms grouped under the mucous membrane as wavy threads, which, on account of their small number, caused no disturbances of health whatever. It is found more frequently in the wild boar and the peccary.

Fig. 36. Egg of Spirotera strongylina.

2. **Gnathostoma hispidum** (Cheiracanthus hispidus); cylindrical worm, 2 to 3 cm. long, thickened at the anterior end, adhering to the gastric mucosa or boring into it, the posterior end projecting free into the lumen. The worm produces intense inflammation of the mucosa, thickening of the gastric wall, dilatation of the stomach, disturbances of digestion, cachexia (Csokor); the parasite was found in Hungarian hogs by v. Ratz and Stroese.

3. **Simondsia paradoxa;** found in England by Simonds, in Hungary by v. Ratz in the stomach of swine; the females, 45 mm. long, were seen inside of cysts of the gastric wall, projecting with their head into the cavity; the males are found free in the gastric contents.

Fig. 37. Ovum from Gnathostoma hispidum (After Ciurea.)

4. **Strongylus rubidus.** This worm was found by Hassall and Stiles in 27% to 75% of all hogs examined in the stock yards at Washington, D. C., and by Crocker and Biester in 60% of 400 healthy appearing slaughter hogs in Philadelphia. Oppermann saw an enzootic outbreak of the disease due to this worm among breeding sows on a farm in Westphalia, Germany. Their infection had taken place in an unpaved runway which was constantly in a filthy condition. In Hungary the disease was observed with an enzootic character by Marek in 6-months-old pigs and was also diagnosed by Kotlán in cadavers from different parts of the country. The worms produced croupous to diphtheroid chronic inflammatory changes in the mucous membrane of the stomach and thereby anemia and chronic wasting away terminating in death. In the author's own observation the disease was characterized by debility and decrease of appetite, later diarrhea and emaciation. The course was chronic. According to Crocker and Biester, the severe and even fatal parasitic changes in the stomach develop only under

the simultaneous action of weakening influences (chronic, debilitating diseases, particularly bronchial pneumonia, insufficient and faulty diet, too great a strain on nursing mothers by the suckling pigs, etc.), which decreases the resistance of the mucous membrane of the stomach, thus allowing an undisturbed invasion of the parasites, while on the other hand even massive invasions are followed only by a catarrhal condition. In all cases one will find in the feces large numbers of elongated oval eggs, on an average 70 microns long with 4 to 16 cells, as Oppermann found in his cases. (Kotlán reports the eggs as 45 microns long and 36 microns wide.)

Fig. 38. Croupous diphtheroid gastritis in a pig produced by Strongylus rubidus. (After Kotlán.)

The anatomical changes (Fig. 38) are found in the fundus and pylorus region of the stomach as moist, mucous or more fibrin-like, frequently also dry, adhering deposits, from the size of a lentil to that of a silver dollar and up to ¼ cm. thick, around which the mucous membrane is intensely red. In and around these deposits, as well as frequently also in the neigh-

boring mucous membrane, the brownish to grayish white worms (5 to 8.5 cm. long) can be found. Without preceding weakening influences, there will be found, according to Crocker and Biester, a catarrhal secretion in varying quantities, while microscopic erosions on the crests of the folds of the mucous membranes may be recognized with a reading glass; also microscopic parasites embedded in the mucous membrane surrounded by rings of degeneration or necrosis in the immediate vicinity.

5. **Physocephalus sexalatus** was found, associated with Spiroptera strongylina, by Foster.

The trail has hardly been blazed for the **treatment** of worms in the stomach of pigs. Under all circumstances it seems advisable to withhold food from 16 to 24 hours before treatment. The administration of vermifuges by mixing them with the feed is irrational (inexact dosage, wasteful). Each pig should be dosed separately and the dose administered in the form of an electuary, pill, capsule, solution or in a fluid mixture (for particulars, see page 237). Oppermann gave a sow twice daily for 8 days a mixture of 30 gm. Glauber salt and sodium bicarbonate (5 to 1), and then 0.05 gm. arsenic with 4 gm. calomel, whereupon, after 5 days, only 2 eggs of Strongylus rubidus could be found in the feces and the animal gained 23 lbs. in weight in 3 weeks. Youatt recommends oil of turpentine (1 coffee spoonful) and sodium chloride with the feed. (According to Hall and Foster, oil of turpentine is unreliable in the pig and may become dangerous by the production of debility, depression of appetite, frequent micturition and nephritis.) According to Foster, drugs similar to those used in stomach-worm disease of sheep (see page 307) should be given in proper doses; furthermore, santonin (0.5 to 1 gm.) with calomel (0.5 to 1.2 gm.) given at a single dose, or, better, in repeated doses at intervals of 3 days. Furthermore, drugs effective against ascariasis (which see) should prove effective. (In mass treatments it is advisable first to test the vermifuge as to its effectiveness.)

As a prophylactic measure the still healthy animals (examination of the feces for eggs if necessary) should be moved to a clean place, the feces removed daily from the hog lot or barn and mixed with unslaked lime or stored in a place not accessible to hogs. The barn should be disinfected by first removing the manure and top layer of soil and then whitewashing the structure.

(e) Parasites in the Stomach of Carnivora.

In the stomach of **dogs** Spiroptera sanguinolenta is found most frequently enclosed in tumorlike formations; it usually does not produce disturbances.

Larvæ of Gastrophilus equi have been found a few times in the stomach of dogs, they were probably swallowed with horse manure and had attached themselves

to the gastric mucosa (Railliet). Ascaris marginata and Tænia may get into the stomach from the intestines.

Ollulanus tricuspis is parasitic in the stomach of **cats.** It is a slender worm one mm. long and is sometimes found in exceedingly large numbers in the catarrhal gastric mucosa; the females, which are provided with three nodules at the tail end, produce live embryos. The latter subsequently wander from the stomach and intestines into various organs (lungs, liver, diaphragm, pleura) where they may subsequently be found encapsulated in cysts of the size of a pin head. The larvæ voided with the feces or the bronchial secretion are found in the muscles of small rodents encapsulated in a manner like trichinæ, and cats probably infect themselves from the meat of mice.

(f) Stomach-worm Disease of Rabbits and Hares.

Strongylus strigosus (Graphidum strigosum) occurs not infrequently in the stomach of rabbits, more particularly of hares; it is present in great numbers and causes anemia, hydremia and ascites. The infection is probably transmitted through the drinking water, in which the embryos leave the eggs which have been discharged with the excrements; the eggs are elliptical, 106 μ long, and are grooved when deposited. In the course of an enzootic of the disease in rabbits in a reserve Abspoel found the inflamed mucous membrane studded in places with hairlike red worms 2 to 3 cm. long, and the mucous membrane perforated like a sieve. Similar changes were seen by Schlegel in a case where numerous rabbits had died.

The usual anthelminthics are to be employed for treatment (see the chapter Tape-worm in Rabbits), and care should be taken to keep the rooms in which the rabbits are kept dry and clean, for the sake of prophylaxis.

(g) Parasites in the Stomach of Fowl.

Chickens. In the glandular and muscular stomach the **Dispharagus nasutus** (Spiroptera s. Filaria nasuta) occurs, a filiform worm, 5-9 mm. long, with two mouth-lips and two skin-bands passing from the mouth-lips and being bent ventrally. The eggs are 38 μ long, oval, with thick shells, they contain a rolled-up embryo which is said to live for a time in a small crab after leaving the egg (Piana). Occasionally the disease produced by this worm occurs in enzootic distribution (Legros, Colucci, Piana, Nelman). Both portions of the stomach may be involved or only one; the glandular stomach is enlarged, its mucous membrane thickened and peculiarly villous in appearance if many parasites have bored into it. The inner surface of the muscular stomach soon becomes rough in consequence of the presence of many parasites, some of which have dug themselves completely into its walls. In some regions the Dispharagus hamulosus is met with, also D. laticeps.

Eber observed in two flocks of chickens in which many had died from D. spiralis the brownish red worms (blood suckers) rolled up in communicating cysts half the size of a lentil at the cuticular edge in the gizzard, partly projecting above the mucous membrane and filled with a clotted mass.

The clinical symptoms are emaciation and listlessness, although the appetite is preserved. The animals perish from debility. The microscopical demonstration of the eggs, in the droppings or in the contents of the crop renders the diagnosis certain.

For treatment Colucci recommends oil of turpentine (0.5 gm. in gelatine capsule, twice daily for several days). The droppings of the affected animals must be rendered harmless and the chicken houses must be kept dry and clean.

Wasielewsky and Wuelker found a tumor in a homing pigeon that entirely filled the glandular portion of the stomach and which upon examination proved to be an adenopapilloma produced by Dispharagus.

Spiroptera pectinifera is a thread-worm, 4-9.5 mm. long, whitish in color; with two conical mouth-lips and behind them two halfmoon-shaped papillae which are denticulated on the concave surface. The eggs contain embryos. The worms live in the muscular stomach, causing ulcers up to a nickel piece in size, with undermined margins and bloody base; in other places they cause brownish deposits on the mucous membrane, consisting partly of blood, partly of worms which have dug themselves into the mucous membrane. The disease may be enzootic and causes many deaths (Nicolas).

The symptoms are emaciation, in spite of good appetite; diarrhea, undigested food being found in the droppings, as well as the worm eggs which can be shown under the microscope. After 4 to 6 weeks, sometimes only after several months, cachexia develops which leads to death. The mortality amounts to 9 to 25%.

For treatment oil of turpentine may be employed or another worm remedy, as in tapeworm. The houses must be kept dry and clean, and the droppings must be rendered harmless.

Centoscudi found **Spiroptera perforans** in a chicken in the fibrinous exudate in a diverticulum of the gizzard the size of a walnut and communicating with the gizzard.

Weidmann observed in a zoological garden numerous deaths in parrots as the result of destruction of the glands in the glandular portion of the stomach, occlusion of the esophagus, rupture of the glandular portion of the stomach and intoxication by Spiroptera incerta, a thread-like worm of which the life history is still unknown. Many birds recover after spontaneous death of the worms. Vermifuges are not effective. The disease can be combated by isolation of the birds found to be infested by microscopic examination of the feces after they have been dissolved in potassium hydroxide and centrifuged.

Ostrich. According to Theiler and Robertson, Strongylus douglasii (Haarworm, Draatworm) caused heavy losses among ostriches in South Africa. It is a yellowish-red strongyle, 4.6 to 5.6 mm. long. Its elongated oval eggs are 59 to 64 microns long and at the time of passage contain embryos some of which have already hatched. After two moltings (at 35.6° C. in 59 hours) the larvæ become infective and go through two more moltings in the animal body.

Resistance. In the droppings the eggs and larvæ resist drying for two years. In droppings on the ground they live for 14 to 17 months, in moist air 15 months, in water 13 months, and in a frozen condition 11 days. Freezing does not influence the hatchability of the eggs, but no larvæ capable of infesting a new host develop under a column of water 5 to 7 cm. high.

Anatomical changes develop in the glandular stomach, the worms penetrating into the deep and partly also into the superficial glands. In old birds they approach the surface of the mucous membrane and attach themselves to it. The mucous membrane becomes thickened in cases of massive invasion.

The **symptoms** consist of emaciation, anemia, chalky precipitates in the droppings and frequently death. Adult birds when well fed tolerate even massive invasions without harm.

Good results have been obtained with the administration of slaked lime and sal ammoniac (for 6-weeks-old birds a teaspoonful, for 9-months-old birds a heaping teaspoonful, and for older birds 14 to 28 grams of the mixture, preferably dissolved in water), after which, for the purpose of facilitating the loosening of the worms, paraffin (according to age 14 to 300 gms.) is given.

For prophylactic measures the following are recommended: Exclusion of young ostriches from pastures that have already been used by older ostriches; intensive feeding of adult ostriches; cleaning and disinfecting the yard for young ostriches after the removal of the top layer of soil to a depth of 7 to 8 cm.; feeding the young ostriches with feed obtained from places where no old ostriches are kept. When old and young ostriches are kept in the same yard, the yard should be cleaned daily. Only artificially hatched chicks or chicks taken from the nest soon after hatching should be placed in the yard for young ostriches. Birds with even only a latent infection should be excluded from the yard and pasture for young ostriches. In order to diagnose such infections, place the droppings in a covered glass jar; in a few days the larvæ can be seen with the naked eye, crawling on the wall of the jar, their number indicating the degree of infestation.

Water-fowl. In the glandular stomach of ducks, geese and swans, the **Dispharagus uncinatus** (Spiroptera s. Filaria uncinata) is parasitic, which is distinguished from the D. nasutus by the wavy course of the skin-bands, and by the presence of a double row of hooks between the skin-bands; it is up to 18 mm. in length. The eggs resemble those of D. nasutus, but according to Hamann the embryos are liberated already in the stomach of the host; after being discharged with the droppings, they are taken up by small crabs (Daphnia pulex) and develop in their body cavity into larvæ of 1.7-2 mm. in length. The infection takes

Among the symptoms, mention is to be made of the diminished appetite, emaciation, sometimes difficult deglutition. In ducks the manifest disease lasts only a short time, the animals refuse food, are feeble and listless, their motions are staggering, the eyes are cloudy as early as the end of the first day; the mouth is opened wide, death occurs after one day's illness, but may be even more sudden.

For prophylaxis, the killing of the affected animals is of importance, also keeping the water-fowl away from suspected stagnant waters which are colored yellowish or brownish if the Daphnia are present in large numbers, especially between June and September (Hamann).

Epizootics caused by **Tropisurus fissispinus** (Tropidocerca fissispina) have been repeatedly observed (Rust, Eber, Grosso, Schlegel) in ducks. The worms (males 3 to 6 mm., females 2 mm. long) are located in the glandular stomach, partly attached to the mucous membrane, partly lying in the glands, and producing ulcerous changes or forming deep blue to bloody brownish-red cysts, each of which contains a bloody brownish-red worm. The more egg-shaped females can be recognized as small red dots in scrapings from the mucous membrane. According to Grosso, the severe digestive disturbances are partly due to an inhibition of the activity of the numerously attacked glands of the stomach. The infestation was transmitted by Daphnia (Gammarus) pulex.

Hystrichis tricolor is a worm, 2.7 mm. in length, provided with hooks at the anterior end, and belonging to the family of the Eustrongylideae. It may occur in the glandular stomach of ducks and may produce severe destruction of tissue. Schlegel reports on ducks which had shown uncertain gait, difficult respiration with open mouth, emaciation, anemia and diarrhea; after death he found ulcers which were as large as a quarter of a dollar, almost perforating; underneath the serosa there were bean-sized nodes in which the parasites were found.

In swans the Hystrichis cygni is found.

Amidostomum anseris (Strongylus nodularis) belongs to the family of the Strongylides and lives underneath the mucous membrane in the muscular stomach, also in the glandular stomach and in the gullet of geese. The male is 10 to 16 mm. long and has a broad trilobate bursa with 2 short spicules. The female is 12 to 22 mm. long. The eggs are an elongated oval, 115 to 120 microns long, thin shelled and contain embryos. According to the observations of Freese, Jost, Reinhardt and the author, the parasite causes disturbances only in young geese or goslings when present in large numbers, while a light invasion seems insignificant even for young geese. Older geese are apparently not harmed even when the parasite is present in large numbers (carriers). The changes in the gizzard are found in the softer places of the mucous membrane adjacent to the strongly cornified ridges, at which place the worms penetrate into the openings of the glands and into the deeper layers of the loosened keratinoid layer; the larger part of the body of the parasite, however, remains on the surface of the mucous membrane. Through their activity the keratinoid layer is destroyed and an inflammation of the mucous membrane without local eosinophilia is produced (Joest). Petechiæ and dirty bluish-gray to blackish-brown discolored places will also be found in the loosened keratinoid layer with some of the worms projecting from its clefts. The corneal layer can be easily detached in the form of hardlike, crumbly, necrotic pieces, whereupon the mucous membrane proper, covered with a layer of densely matted worms, becomes visible. The worms are attached to the surface of the mucous membrane proper.

By sucking blood, by excreting toxic substances, by producing disturbances in the activity of the stomach, and probably by favorably influencing bacterial infection, the worms cause a rapidly progressing emaciation and debility in spite of a good appetite which is sometimes maintained almost to the end. Death takes place in from 3 to 5 days. A clinical diagnosis depends upon a microscopic demonstration of the eggs and embryos in the droppings.

Treatment does not promise results. For prophylaxis, the young geese should be separated from the adult birds, kept away from dirty barns, and particularly their quarters carefully cleaned daily.

29. Worms in the Intestinal Tract. Helminthiasis.

Action of the Parasites of the Stomach and Intestines. Contact with the mucosa, adhesion to it, boring into it, produce an irritation, which, according to the number of worms, may cause a circumscribed or a diffuse catarrh or even an intense inflammation. Worms armed with hooks may penetrate more deeply into the mucosa; they may even perforate the intestinal wall and cause fatal peritonitis.

Great numbers of larger worms may unite in lumps and may narrow or obstruct the intestinal lumen. On the other hand individual worms may penetrate into a duct opening into the intestines, especially into the bile duct; they may occlude it and prevent the discharge of a secretion or excretion; they may exceptionally get from the intestine into the stomach, esophagus, pharynx, buccal cavity or larynx, where they cause irritation and, in exceptional cases, stenosis.

By withdrawal of nutritive material intestinal worms, when present in larger numbers, cause emaciation, eventually also anemia of the host; in the production of the latter the toxins (leucomaines or ptomaines [Linstow]) of the worms play an important rôle.

Poisonous substances have been demonstrated in many helminthes. They produce destruction of the red blood corpuscles, reduction of hemoglobin, anemia with poikilocytosis, the appearance of nucleated red blood corpuscles, including megaloblasts and an eosinophilia of the blood. They also produce an increased decomposition of the proteids of other organs.

They have an unfavorable influence upon the glands with internal secretion (adrenals, thyroid, etc.), and partly in this manner, but also directly, they may produce nervous symptoms (excitement, convulsions, paralysis), which disturbances were formerly erroneously ascribed to reflex actions. Under certain circumstances even a verminous anaphylaxis may be produced by the periodic absorption of specific worm proteins from dead worms or worm toxins. (In the motor ganglia of the spinal column of guinea pigs sensitized with extracts of Ascaris megalocephala, Taenia plicata and Schlerostomum equinum, Rachmarrow found changes similar to those in horse-serum anaphylaxis.)

By producing numerous injuries to the mucosa of the intestinal tract, many worms favor bacterial infection and in this way produce severe inflammation, general disturbances and sudden nervous symptoms. By migration of their larvæ from the skin or the intestines, many of them often carry pathogenic bacteria along with them (proved experimentally by de Blieck and Baudet for Strongyloides westeri). Even a protozoal infection is sometimes favored by their presence in the intestines (according to Smith, the carrying along of the causative agent of enterohepatitis meleagridum by heterakids).

Diagnosis of Parasitic Diseases of the Stomach and Intestines. The different representatives of the parasites of the stomach and intestines are very frequently found in domesticated animals. Their presence is always to be assumed when, with or without symptoms of a gastrointestinal catarrh and in spite of an unchanged or even increased appetite, emaciation and poor development (particularly in young animals) takes place, and when in flocks or among animals kept in enclosures

similar conditions develop. If under such condition no macroscopically discernible parasites have been passed, or if examination of the feces for parasites in the barn or at the sleeping quarters yields negative results, and if a post-mortem examination of a dead animal has not cleared the situation, a trial administration of laxatives or vermifuges to a few animals in the flock followed by the passage of a few parasites should render a decision possible. In a majority of cases the presence of parasites in the stomach and intestines can be ascertained during the life of the animal only by microscopic examination of the feces. With sufficient practice and the use of proper methods, the degree of infestation can also be approximately determined. It should be borne in mind, however, that in a given case the detection of animal parasites in the gastrointestinal tract does not necessarily prove the parasitic nature of the disease, since parasites present in only small numbers do not produce a recognizable disturbance of health. The external symptoms of the disease must, therefore, be in harmony with those occurring in the different parasitic diseases, while on the other hand the number of parasites and of their larvæ and eggs must also be considered.

The degree of the disturbance varies, on the one hand, according to the species, number and location of the intestinal parasites, and on the other hand according to the sensitiveness of the diseased animal. Large worms, particularly when they are provided with hooks, are, as a rule, more dangerous, while on the other hand large numbers of small worms (Taenia echinococcus, Dochmius) also may produce severe disturbances. The sensitiveness is also influenced by the species and individual character of the suffering animal, particularly in horses and dogs.

Microscopic Detection of Animal Parasites and Their Eggs. By reason of its simplicity and reliability, the agitation method of Pataki is best suited for practical purposes. Samples are taken from different places of the pellets of sheep, goats and rabbits, from a fecal ball of a horse, or from partly desiccated feces of a dog, and agitated with water in the proportion of 1 to 3 (approximately). Soft fecal masses of the consistency of normal cattle feces are agitated with water in the proportion of 1 to 2, and thin feces in the proportion of 1 to 1, while liquid feces are used as such and treated like those agitated with water. The samples are placed into a conical glass or into some other vessel and thoroughly agitated with a wooden stick or glass rod until a uniform suspension is formed. Immediately following, a small sample is taken up with a pipette, a drop of which is placed on a slide and covered with a cover glass, under which the mixture spreads out in a uniformly thin layer. Thus prepared, the specimen is searched systematically. Should the search last for some time, the suspension must again be thoroughly agitated before another sample is taken. In counting the parasite eggs or larvæ for the approximate determination of the degree of invasion, and at the same time the approximate determination of the number of parasites in the hosts, it is preferable to count for cattle 10 preparations, for other species 5, in order to get a more exact average. In the method by Leichtenstern, which is also simple, a sample of feces is mixed with 2 to 3 times its volume of water, placed in a shallow dish and allowed to settle for a short time. A small portion of the sample is now taken up with a platinum loop and placed upon a slide. The results are not so good as with the agitation method.

As an improved method, especially for scientific investigation as well as for spotting carriers harboring only a few worms or protozoa (latent invasion), the sodium chloride enrichment method may be used as well as the method of Bass and Lewis, which is fundamentally similar to it. The sodium chloride enrichment method introduced by Kofoid and Barber, improved by Fuelleborn, and especially by Noeller and Otten and by Hohmaier and Taube, is exceedingly useful and very simple for the examination of the feces of the different species. To a sample of feces the size of a pea to that of a small egg (cattle), concentrated sodium chloride solution is added drop by drop while stirring with a wooden spatula until a fluid paste is obtained. It is then placed upon a wire sieve with 0.2 mm. meshes and concentrated sodium chloride solution poured over it and stirred to separate the coarse particles. The proportion of feces to salt solution should be as 1:8-25 (stronger dilutions are insignificant, with only a small sample of feces even advantageous). The strained salt solution is placed into an Erlenmeyer flask, the walls of the neck of which run parallel, or into any other suitable vessel (according to Fuelleborn, only cylindrical), which is now closed with a well-fitting stopper and set aside from 25 to 90 minutes. The specifically lighter oocysts of coccidia, as well as the eggs of most roundworms (Ascaris, Heterakis, Sclerostomum, Ancylostomum, Oxyuris, Echinorrynchus) and many tapeworms (of the horse and cattle), will be found in the superficial layer of the sodium chloride solution without the admixture of foreign matter (with feces from carnivorous animals a creamy layer of fat and protein remnants will be found on the surface), from where they may now be obtained with a bent platinum loop of 0.3 to 0.4 cm. diameter, and even their further development followed up. With the rapid sodium chloride method the fecal suspension, after allowing the coarser particles to settle for a few minutes, is distributed into centrifuge tubes, centrifuged for a short time and the surface layer examined immediately.

With the glycerin method by Vajda the feces are diluted with water (under normal conditions to feces of horses and hogs double their volume of water, to that of sheep, goats and dogs three times their volume of water, to thin samples the amount of water must be decreased correspondingly, while no water is to be added to liquid feces) and then thoroughly mixed with glycerin (specific gravity 1.25) in a wide test tube. For the detection of eggs of Strongylus, Oxyuris, Dochmius and Oesophagosomum and of coccidia the glycerin is added to an equal volume, for Ascaris megalocephala 1½ volumes, and for other intestinal worms 3 volumes. The mixture is allowed to stand from half an hour to an hour, or it is centrifuged and then also allowed to stand for a short time. By simply touching the surface of the liquid with a dry glass rod, one will always find the eggs most numerous in the drop of liquid thus removed. In examining large samples of feces of the horse or pig, the sample is mixed with double its volume of water, strained through a cheesecloth, taking care to express all the liquid, which is now centrifuged for 30 minutes. The water is then poured off up to within 1 to 2 cm. of the sediment, to which now is added, for feces from horses, 1 to 1½ volumes, for feces from hogs 3 volumes, of glycerin; the whole is mixed thoroughly, centrifuged again for 15 to 20 minutes and allowed to settle for a short time. In the brownish, creamlike layer on the surface the eggs of the parasites may sometimes be found in large numbers. By fastening a piece of filter paper over the mouth of another test tube and touching the surface of the centrifuged mixture with this, the worm eggs may be removed in large numbers and preserved in a closed vessel for months without being damaged. Some eggs can even be seen with a reading glass on a slide. Only the eggs of Distomum are injured by the glycerin (bursting of the shell after a few minutes).

The cultural method for the detection of larvæ of parasites is also suitable in general practice, as, for instance, the method recommended by Theiler and Robertson for the diagnosis of stomach-worm disease (see page 326, 336), or the method practiced by Looss for the detection of Ancylostoma larvæ. According to the latter method the fresh, soft feces are mixed into a thick paste with an equal volume of finely pulverized animal charcoal (the charcoal acts as a carrier of oxygen and prevents decomposition), and the mixture is transferred into Petri dishes or other containers and placed into an incubator at 25° to 30° C. for 5 to 6 days. During this time numerous larvæ will develop inside the mixture, which has now become dry on the surface. After a few cubic centimeters of warm water have been added and the mixture again placed in an incubator for 10 to 20 minutes, the larvæ will collect in the water in large numbers and may be detected with a reading glass, or, better, by centrifuging the

water and pouring off the supernatant fluid, whereupon they will be found in the sediment of the few remaining drops.

With the method of Adelmann and of A. Albrecht for the differentiation of the larvæ of the different species of Sclerostomum, a fecal ball of the horse is placed into a glass vessel and this protected from the light by wrapping it in a piece of black paper, covered tightly and placed near a stove. After 2 days actively moving embryos can be seen in the feces (Adelmann). Albrecht preserves the fecal ball, protected from light and drying out (covered loosely), for 8 to 14 days (in summer 5 to 8 days), then pours water or saline solution over it until it seems thoroughly soaked and some water has collected at the bottom of the vessel. In the course of a few hours and with favorable illumination one will see in the water, even with the naked eye, very small worms which frequently collect in small balls. Sometimes the detection is possible only after carefully decanting or centrifuging the water.

For the detection of coccidia the flakes of mucous that are admixed with the feces are best suited. Sometimes they are also found in the clots of blood. They are most easily found when these admixtures to the feces are, without further treatment, examined under a cover glass with a medium magnification. The sodium chloride method is especially successful. For staining smears Zueblin recommends Lugol's solution.

For the detection of amebæ fresh smears are made from the mucopurulent flakes in the feces, if necessary diluted with saline solution, or, better still, this mixture is examined in a hanging drop. The amebæ will then be recognized as bright, shining, actively moving discs with strongly developed pseudopodia.

According to the observations of Noeller, the Heidenhain method of staining, fixed moist cover-glass smears from the feces can also be used for the detection of protozoa (also when already encapsulated) of animals. With this method the smears are fixed with corrosive sublimate mixtures or picric acid mixtures; the corrosive sublimate is then removed with tincture of iodine, the picric acid with 70% alcohol, and the cover glasses are placed in 40% freshly prepared and filtered iron-alum mordant for 1 hour at 37° C. in an incubator (if necessary 12 to 24 hours at room temperature) and then washed in a light stream of hydrant water for 1 to 2 minutes. The smears are then placed in the customary Heidenhain staining solution that has ripened for at least several days but must not be too old, for 1 hour at 37° in the incubator (if necessary 12 to 24 hours at room temperature), then washed in running hydrant water and covered with 2% iron-alum solution, in case of amebæ, for 3½ to 4 minutes, for intestinal flagellates, for 1½ to 2½ minutes, and for infusoria, for 5 to 6 minutes. The iron-alum solution is then poured off, the smears thoroughly washed in hydrant water, passed through alcohol to xylol and embedded in Canada balsam or in larch turpentine (Venetian turpentine) directly from 96% alcohol. A weak counterstain with eosin (1 to 2 minutes with a 1% aqueous solution) is advantageous when the differentiation has been carried too far. When examining simultaneously for ameba and flagellates 4 smears treated alike with the same mordant and stained may be differentiated in series (1½, 2½, 3½, 4½ minutes) and embedded 2 each for the flagellates and the amebæ. For staining cysts of amebæ the mordant and stain may each be allowed to act for 3 hours in the incubator.

A. Tapeworms. Cestodes.

Tapeworms are bandlike colonies of hermaphroditic individuals arranged in adhering segments. The anterior segments of each chain (strobila) forms the so-called head (scolex), which continually forms new segments asexually, by longitudinal growth and transverse segmentation, that is, individuals which adhere to each other. The more distant the segments are from the scolex, the larger, wider and longer do they get. Those situated caudally show more distinctly the sexual organs, which attain their full development in the last segments of the band (strobila). The mature segments are also called proglottides. There is a newer tendency to look upon the tapeworm body as

an individual which separates portions of its body, by constriction, during its continued growth in length. The individual segments are hermaphroditic; the uterus usually contains numerous eggs, and it shines through the external layer of the proglottides; its characteristic shape may be utilized for determining the species. The sexual openings or pores are on the margin of the individual segments in Tænidæ, and on the flat surface in Bothriocephalidæ.

The segments are formed by a double layer of connective tissue; the external layer contains small granules of lime. The nutritive material of the intestinal contents of the host penetrate into the body of the worm through small openings of the segments. The excretory products of the worm are voided through a system of canaliculi contained in the lateral portions of the segments. The canaliculi terminate in an opening on the posterior margin of the last segment.

The **development of tapeworms** occurs as follows: After the ova, which are voided with the segment, have gotten into the stomach of a proper host, their shell is dissolved and the free embryo (oncosphere) perforates the wall of the stomach or intestine, and gets actively or passively into various organs; in case it comes from a tænia it changes into a bladder-worm or cysticercus; while the embryo of Bothriocephalidæ forms a band-like pleurocercoid. If the cysticercus or pleurocercoid subsequently gets into another proper host, the scolex becomes extruded, the bladder or the body of a pleurocercoid is digested and the free scolex or pleurocercoid wanders into the intestine, attaches itself to the mucosa and the development of segments begins. Embryos are formed in the ova after auto-fertilization. From time to time, shorter or longer portions of the worm become detached and are voided with the feces (T. echinococcus consisting of three or four segments is voided completely), the segments remain outside on the ground, on straw, or grass. They remain alive for a few days, show even some motion, then they die, the ova, however, are protected by a thick shell, and remain alive much longer, provided that there is some moisture. (According to feeding experiments by Leuckart, Roell, and Gerlach, up to 5 weeks.) By direct sunlight they are killed in a few days (Leuckart), while the power of further development is lost by drying out in less than 2 weeks. Bothriocephalidæ deposit their ova in the intestinal tract of the host.

The morbid effect of tænia is due to a withdrawal of nutritive material, also to their poisons (Linstow); serious disturbances of health are observed only in the presence of numerous tapeworms. (Jerlov often obtained positive complement-fixation reaction in man suffering from tænia or bothriocephalinæ with alcohol-ether extracts of tapeworms.)

(a) Tapeworms in Horses.

Etiology. Three hookless tapeworms occur in the horse;

their segments are broad and short (trapezoid), often misformed, with the sexual pores located on the same side. The immature stages are not known (they are probably to be sought in small mollusks, insects and worms in the pastures).

1. **Anoplocephala (Taenia) plicata:** 10-80 cm. long, flat tapeworm with comparatively large (4-6 mm. wide) square head, supplied with four strong suckers. The ova are round or cubical (50-60 μ long). The tapeworm is found rarely in the small intestine of horses, even less often in the stomach.

2. **Anoplocephala (T.) perfoliata:** 2.5-8 cm. long and 3-15 mm. wide; found in the small intestine, exceptionally also in the stomach. Head blunt, 2 mm. thick with well developed suckers, behind the head on the upper and lower side two rounded flaps. The ova are polygonal. It lives in the small bowel and in the colon, exceptionally in the stomach. Of all tapeworms it is found most frequently in horses, especially in Russia. The eggs are polygonal, 65-80 μ long.

3. **Anoplocephala (T.) mamillana:** Only 1-5 cm. long and 4-6 mm. wide. The oval suckers found on a polygonal, small head are provided with a central furrow-like depression. The ova are elongated. Is found in the duodenum, jejunum and ileum.

Tapeworms are comparatively rare in horses; A. perfoliata is the most common among them. The cysticercus of these tapeworms and the manner in which horses are infected are unknown.

Anatomical Changes. If the intestine contains many tapeworms, its wall bulges out here and there, and the worms are found in these sacculations. The walls of the latter may rupture later on, and this will lead to fatal peritonitis (Mégnin, Perroncito, Waldmann).

Symptoms. Tapeworms rarely lead to any marked disease which then will manifest itself in chronic digestive disturbances, possibly in emaciation or anemia, occasionally in symptoms of colic. In a stud in Hungary an enzootic infestation with tapeworms in 1 to 2-year-old colts at pasture is observed every year (15 to 20 per cent) by Broskó. (Hendricks and Wilkinson observed enzootics in foals, due to A. plicata; three foals of Hendricks' cases died.) Tænia can only be assumed to be the cause of such disturbances after the proglottides have been seen in the voided feces.

Treatment. The expulsion of tapeworms can be accomplished fairly easily with laxatives, such as aloe (20-25 gm.); tartar emetic (10-12-20 gm. pro dosi or in fractional doses 30 gm. per day). More reliable is the effect of oil of turpentine (100.0 gm. with 500 gm. of castor oil), arsenic (in increasing doses from 1 to 3 gm. per day for 1 to 2 weeks), calomel (4-6.0 gm.), areca nut (daily 2-3 times two tablespoonfuls, until one quart has been given), cinna leaves or rhizoma filicis maris (50-100.0 gm.)

(b) Tapeworms in Cattle.

Etiology. The tapeworms found in the intestinal canal of

cattle belong to the anoplocephalic tæniidae having heads without hooks, large suction-cups and broad, short segments. The larval forms, and therefore the evolution as well as the mode of infection are unknown.

Fig. 39. Moniezia expansa (mature segments).

1. **Moniezia (Taenia) denticulata:** Normal length 25-80 cm., but may be 170 cm.; the head is somewhat wider than the first segments and possesses suction cups directed forward. According to Railliet this tapeworm does not occur in ruminants, but only in rabbits and is identical with Ctenotaenia Goezei. The eggs are 48 μ long, rounded or polygonal.

2. **Moniezia (T.) expansa:** The longest of all tapeworms, not infrequently 10 meters long. The head is small (0.5-0.8 mm.), pear or club shaped with four oval suckers with slit-like depressions. The male and female genital pores are separate on the lateral margins of the segments. The ova are like those of the preceding species, 50-90 μ long. This tapeworm is the most frequently found in cattle.

3. **Moniezia (T.) alba:** Not more than 2.5 meters long; the head comparatively large (1.4 mm.) with large oval suckers, ova cuboidal. Sometimes the M. planissima and M. Bendeni are found. Also the M. conjugens, M. latifrons, Stilesia centripunctata and S. globipunctata (a variant is found in the dromedary); Thysanosoma giardi.

The enumerated worms occur in the small intestine; their mode of development is unknown; M. denticulata is the species most commonly found in cattle.

Symptoms. M. denticulata only sometimes causes more considerable disturbances, such as digestive disturbances, emaciation, anemia and bloating (Haubold & Trülsen); exceptionally, epileptiform attacks (Fumagalli, Eggmann). These worms usually do not disturb the health of cattle.

Treatment. The same drugs as those recommended above for the horse may be utilized.

(c) Tapeworms in Sheep and Goats.

Etiology. In the intestinal tract of sheep are found somewhat frequently only the **Monieza (Taenia) expansa,** and the **Moniezia (T.) alba.** More rare are Thysanoma ovillum (Tænia ovilla) according to Perroncito, more frequent in Italy; according to Lungwitz, less so in Germany; Th. actinoides very frequent in North America; Moniezia Benedeni, M. trigo-

nophora, Stilesia centripunctata, St. globipunctata and others.

In goats the Moniezia expansa and the M. caprae are found.

From a practical standpoint the most important is Moniezia expansa, because in summer it may occasionally cause numerous infections in lambs and yearlings, exceptionally also in older sheep, and may even cause numerous deaths (so-called worm epizootics). Lambs born during the winter, most frequently fall victim to this disease if they are driven to the pasture in spring or at the beginning of summer, during moist weather. Although more rarely, the disease may come on during stall feeding, and lambs sometimes sicken when they are still very young. Spinola found tapeworms 10 meters long in a lamb of four weeks; this points to a very rapid development of the tænia since infection can have occurred only after the birth of the lamb.

Symptoms. The symptoms of infection are primarily only disturbances of digestion that are not very marked in character; later on some animals of a herd are less lively, they separate from the herd, become emaciated and remain stunted in development. The mucosæ of such animals are found pale, the wool is dry and can be pulled out easily. The animals occasionally exhibit symptoms of colic, strain without voiding any feces and then run away with their tail elevated. Later on the feces become mushy or even thin fluid, and in them may be recognized the yellowish white, motile proglottides. Occasionally obstruction of the small intestines by masses of worms occurs, death then usually ensues rapidly, even before the emaciation has been fully developed. (Blumenberg saw numerous fatal cases due to intestinal obstruction of this kind.)

Treatment and Prophylaxis. It is advisable to give green feed to the animals for several days before the treatment proper is instituted, also pine needles or juniper berries. The following anthelminthics are to be recommended: Oil of turpentine alone or mixed with hartshorn oil ($\overline{aa}$ teaspoonful), extractum filicis maris æthereum (5-10 gm. with oil), (Ransom and Hall found it to be unreliable), kamala (4-6.0 gm., recommended strongly by Hartmann upon the results of comparative experiments), picric acid (0.1-0.2 gm.) and picrate of potash (0.5-1.5 gm. in pills); except kamala, these drugs should be combined with, or followed in 2 to 3 hours by laxatives (0.2-0.5 tartar emetic, castor oil in tablespoonful doses). This course is to be repeated after 1 to 2 weeks.

Prophylaxis consists in keeping the sheep, especially the lambs, away from infected pastures.

(d) Tapeworms in Dogs.

Etiology. The tapeworms which are parasitic for dogs

mostly belong to the tæniidae; but sometimes Bothriocephalidae are noted.

1. **Taenia serrata**: 0.5-2.0 meters long. Head small, supplied with a wreath of hooks 34 to 38 in number. Segments (Fig. 43) about 400, in the middle of the chain square, more distally they are elongated, rectangular (8-10 mm. by 4-6 mm. wide); the posterior margin projects laterally, so that the lateral margin of the chain is serrated. The uterus has 8-10 branches on each side. Ova oval 36-40 μ long; 31-36 μ wide.

The larval stage, Cysticercus pisiformis develops in the omentum and mesentery of rabbits and hares and with these organs usually gets into the stomach of hunting dogs. After two months mature proglottides are then voided. Rabbits and hares infect themselves from plants contaminated with the feces of dogs.

Lesbre found Cysticercus pisiformis in the brain of a dog infected with T. serrata, where it must have gotten by auto-infection (Friedberger & Fröhner).

2. **Taenia marginata**: (Fig. 42.) 1.5-2.00 meters long. The small head is provided with a double wreath of hooks (30-44) and has four round suckers. The posterior wavy margin of the segments overlaps partly the following segment, the mature proglottides elongated, 14-16 mm. long, 5-7 mm. wide; uterus on both sides provided with 5-8 times dividing branches. Ova somewhat oval, diameter 31-36 μ.

This Tænia develops from Cysticercus tenuicollis, which is found on the peritoneum, the capsule of the liver, exceptionally also on the pleura and the pericardium of herbivora, particularly of sheep and hogs. Butchers' dogs are particularly exposed to the infection, these animals void mature segments 4-5 months after infection.

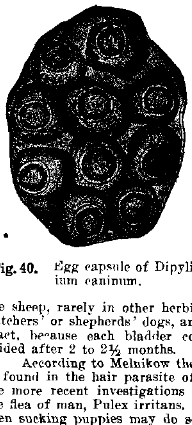


Fig. 40. Egg capsule of Dipylidium caninum.

3. **Taenia coenurus: (Fig. 44.).** It is rarely longer than 40-60 cm., head small, with four suckers and a wreath of 22-23 hooklets. The segments of the median portion are square, the caudal segments elongated; 8-16 mm. long, 3-4 mm. wide. The mature segments are like cucumber seeds. The uterus has 13-25 branches on both sides, the ova are somewhat elongated with a diameter of 31-36 μ.

The larvæ of Cœnurus cerebralis develop in the brain, occasionally also in the spinal canal of the sheep, rarely in other herbivora. These infected parts are frequently eaten by butchers' or shepherds' dogs, and these then develop several Tænia in their intestinal tract, because each bladder contains numerous proscolices. Mature segments are voided after 2 to 2½ months.

According to Melnikow the cysticercus of this Tænia (Cryptocystis trichodectes) is found in the hair parasite of the dog, Trichodectes latus canis, and according to the more recent investigations of Grassi in the dog flea Pulex serraticeps, also in the flea of man, Pulex irritans. Dogs infect themselves by ingesting these parasites; even sucking puppies may do so (parasites 2.5 cm. long were found in a puppy 10 days old, and mature Tænia in puppies 1 to 2 months old).

4. **Taenia echinococcus.** (Fig. 45.) Three or four segments, not over 5 mm. long. Scolex provided with four suckers and a rostellum of 28 to 50 small hooks, arranged in two rows. The second and third segments are incompletely developed and the fourth only is sexually mature. The embryo remains alive in the ovum when desiccated for 10 or 11 days, in water 10 to 16 days. It resists freezing up to 4 months (Dévé).

This Tænia develops from Echinococcus polymorphus found in the internal organs, preferably the liver and the lungs of ruminants, hogs, also of man. After the ingestion of such infected organs by dogs there develop usually numerous Tænia in the intestines of these animals, since every bladder ordinarily contains several hundred protoscolices. Tænia develop from these in 6 or 7 weeks. Different species of Tænia echinococcus are developed from Echinococcus cysticus and E. alveolaris, according to claims of Ostertag, Müller, Mangold and Possell.

5. **Taenia serialis**; 45-72 cm. long; the rostellum rather prominent, with a double wreath of hooks; the segments are much like those of the T. coenurus, only that they are smaller and that their posterior border is more prominent. The evolution is similar to that of the T. coenurus, with the difference that the larval form, the *Coenurus serialis*, lives in the connective tissue of several organs in rabbits and may form daughter-bladders.

6. **Taenia Krabbei**; up to 1 m. in length; head very small; rostellum provided with 26-34 hooks; only the last segments are elongated, the others are more broad than long; the genital pore is very prominent. The larval form is the Cysticercus tarandi which lives in the muscles of the reindeer.

Fig. 41. Dipylidium caninum.

Fig. 42. Taenia marginata.

Fig. 43 Taenia serrata.

Fig. 44. Taenia coenurus.

Fig. 45. Taenia echinococcus. On right, natural size; on left, enlarged.

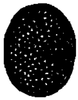


Fig. 46. Ovum of Bothriocephalus latus.

7. **Dipylidium caninum (Taenia cucumerina)** (Fig. 41) is 10 to 40 cm. long, 3 mm. wide at the utmost. The small head terminates in a rostellum which can be projected and withdrawn between the poorly developed suckers. The rostellum is provided with about 60 hooks, arranged in four rows. The mature segments are pale red, shaped like a pumpkin seed, and show a sexual pore on each side. Several ova are united to a sticky mass, a so-called cocoon or egg capsule (Fig. 40).

The cysticercoid (Cryptocystis trichodectis) lives, according to Melnikow, in the abdominal cavity of the dog louse (Trichodectis canis); according to Grassi and Sonsino, in the dog flea (Ctenocephalus canis), as well as the flea of man (Pulex irritans), the larvæ of which become infected with the embryos of the taeniae and then develop into the cysticercoid in the flea (Joyeux). Dogs become infested by taking up these parasites, which can take place while still nursing (taeniae 2.5 cm. long were found in dogs 10 days old and sexually mature taeniae in dogs 1 to 2 months old).

8. **Mesocestoides lineatus** (T. lineata); 30 to 50 cm., sometimes 2.5 meters long. The body somewhat resembles Dipylidium caninum. The sexual pores are, however, found in the middle of the segments. The immature stage is, according to Kuenstler and Gineste, similar to the cysticercoid of Dithyridium elongatum.

In regions where dogs have occasion to eat fish they also harbor Bothriocephalus (seen in Hungary by v. Ratz). The fish may harbor the Plerocercoids, which are the larval forms of the Bothriocephalidae.

Their most common representative is the **Bothriocephalus latus**, which is frequent in certain regions (in the provinces of the Baltic sea, also around Lake Geneva). It is found in man, and Janson noted it in 50% of the dogs in Japan. In dogs its length is 2-7 meters; its head is shaped like a lancet, with two longitudinal grooves; the mature segments are 4.5 mm. long and up to 2 cm. wide; on the plane of the mature segment a rosette-shaped uterus is visible, the opening of the uterus being in the middle line of the plane. The eggs (Fig. 46) are brownish, oval, 68-70 μ long and 44-45 μ wide, they have a cover which is distinctly visible after the addition of sulphuric acid; the eggs are discharged, while in the grooved stage, through the uterine opening into the intestinal canal of the host. If they get into water, a ciliated spheroidal onkosphere develops with six hooks, which are taken up, according to the investigations of Janicki and Rosen and of Galli-Valerio, by the small crustacea Cyclops stremus and Diaptomus gracilis, in which the ancosphere leaves its eggshell and develops into the procercoid in the musculature and liver of these crustacea. When these crustacea are consumed by certain fresh-water fish (pike, burbot, perch, trout, grayling and others), in the muscles of which it develops into the worm-like, band-shaped pleocercoid. After the final host has swallowed these fish, the tænia develops rapidly; its last segments are mature already in four weeks.

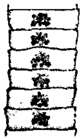


Fig. 47. Bothriocephalus latus (mature segments).

In Iceland and Greenland the Bothriocephalus cordatus and the B. focus are found, in Italy the B. spiratus.

Concerning the **occurrence of various Tænia** in dogs, the following may be said:

Dipylidium caninum is found most frequently, because dogs infect themselves easily by the fleas and lice found in their coat. Butchers' dogs infect themselves easily with Tænia echinococcus, T. marginata and T. cœnurus; shepherds' dogs particularly with T. cœnurus; hunting dogs with T. serrata. The frequency of the occurrence of these tæniae also depends, a good deal, upon external circumstances. Tapeworms in dogs are rarer in large cities with well regulated slaughter houses, with strict meat inspection, and more common in the country where the supervision is less rigid. Young dogs are more susceptible to infection than older animals.

There may be several species of Tænia in one host. The number of parasites also is very variable. Several thousand individuals of the small T. echinococcus may sometimes be found in a single host; Dipylidium also sometimes occurs in several hundred individuals (Krabbe found in one case 2,000).

found in 24.1% Tænia, in 2% Bothriocephalus. Wigdor in America found, in 1,000 dogs, Tænia in 15.2%. Dipylidium in 3.6%. Bouin in south Morocco found,

in 71 dogs, Tænia echinococcus in 14%, T. serrata in 2.8%, T. coenurus in 7%, T. marginata in 9.85%, and Dipylidium in 50.7%. In 200 dogs examined by Deffke in Berlin, he found Tænia in 108 (52%). Zschokke in Zurich found Tænia in 34%, Krabbe in Copenhagen in 65% of the dogs, while in Iceland more than three fourths of the dogs are considered as being infested (especially T. echinococcus is found with relative frequency [30%] and has been introduced to Friesland, Mecklenburg and Normandy by whalers). In Montpellier Blanc and Hedin found 18 out of 25 (72%) dogs infested with T. echinococcus; of 10 dogs running at large 8 (80%) were found to be infested. Meyer reported a significant increase in the number of cases of T. marginata (from 4-5% to 26%) and a multiple occurrence of T. echinococcus (3% of all animals examined) in dogs in Leipsic as a result of the numerous secret slaughterings during the last years of the war and during the years immediately following the war.

Anatomical Changes. The presence of many tapeworms usually causes acute or chronic intestinal catarrh; small hemorrhages also occur in the intestinal mucosa. The intestinal villi are sometimes elongated to 3 or 4 times their normal size, and may form a tunnel over the tænia adhering to the mucosa; the connective tissue is increased, the tubular glands, however, which have been pressed apart, are atrophic. T. echinococcus commonly causes the most intense changes, however, dipylid. caninum sometimes causes croupous inflammations. The lumen of the intestine is very exceptionally narrowed by the pressure of Tænia or completely obliterated. It is claimed that T. serrata may cause perforation of the intestinal wall (Cadéac, Railliet).

Symptoms. Disturbances of health are rarely observed in spite of the frequent presence of tapeworms in the intestinal tract of dogs, and only when the worms are present in large numbers. In general, the symptoms are like those of chronic gastro-intestinal catarrh. The variable appetite, sometimes enormously increased, is generally noticed; it stands in sharp contrast to the emaciation which is evident. Intestinal pains are observed particularly in the presence of T. echinococcus and Dipylidium; they manifest themselves in restlessness, groaning and snapping towards the abdomen; sometimes there are severe nervous symptoms, like dizziness, local or general convulsions, paretic conditions, complete listlessness, especially after intestinal obturation by masses of worms or after the development of enteritis. The sick dogs are also said to exhibit an inclination to bite, dropping of the lower jaw, hoarseness of voice, wild and dull look, symptoms similar to those in rabies. It is claimed that t. echinococcus, when thousands of individuals are present, irritating the mucosa with its hooklets, particularly gives rise to these symptoms (Röll). Cadéac has seen attacks of maniacal excitement in two cases of T. serrata infection, and Fröhner in Dipylidium infection. Liénaux saw multiple hemorrhages caused by the presence of numerous individuals of T. echinococcus. Dobbertin showed that dogs with tapeworms have a leucocytosis of the blood, due to an increase of eosinophilic leucocytes.

One may find proglottides in the feces, in vomited matter

or in the region of the anus; ova of Bothriocephalus are found in the feces, occasionally also ova of tænia. Laxatives ought to be given in doubtful cases, because these generally expel segments of tænia if present; however, T. echinococcus is frequently overlooked on account of its small size.

Treatment and Prophylaxis. After starving the animal for one day and cleaning out the intestine by mild laxatives or infusion of water, the vermifuges are administered. In order to prevent vomiting the animals should first receive some mucilaginous soup. It is sometimes necessary to introduce the vermifuge with the stomach tube, after the stomach has previously been anesthetized; for this purpose one may use a mixture of 0.1 gm. of cocaine, 1.0 of a 0.1% sol. of suprarenin with 10.0 aqua amygdal. amar. (10-30 drops), or anesthesin (0.5 gm.) (Uebele). More rational is the method of preliminary treatment advocated by Hoelt and Seibold, particularly a milk diet lasting for several days until the feces become quite soft or liquid. On the morning of treatment only one-half of the milk ration is allowed, and half an hour to 2 hours later the vermifuge is administered in pills or capsules. The vermifuges to be used are: Kamala (2-8 gm. in milk, honey or syrup), the last, besides being a reliable anthelminthic, acts as a laxative. Extractum filicis maris (0.5-5.0 gm.) in pills, or better in gelatine capsules; one to two hours later, 30-50 gm. castor oil must be administered, or the drug may at once be applied in castor oil (according to Hall and Wigdor, castor oil or calomel administered simultaneously or 1 hour later greatly diminishes the toxic effect of the extract without interfering with its effects upon the tapeworms). Gmeiner recommends as very effective filmaron (0.2-0.4-0.7-1.0 gm. in gelatine capsules or as oil of filmaron); when this drug is used the animals should not be starved; this precaution is necessary to prevent poisonous effect of the drug; one hour after its administration castor oil should be given. Flores cusso (3-5.0 gm. in milk or water are given every hour, repeated 2-6 times); areca nut (5-10 gm.) with butter in keratinized pills; if there is no spontaneous defecation within two hours, castor oil is to be administered; according to Schiel the dosage is as follows: 10 gm. kamala and 20 gm. areca nut are rubbed up with cocoa butter into 25 pills and these are coated with keratin and given in doses of 6 to 15 pills at a time. Regenbogen recommends Bengen & Co.'s worm capsules; these contain 1 gm. areca nut and 1 gm. of kamala and castor oil (5 pills for large, 1 pill for small dogs). Cortex punicæ granati (5-50.0 gr.) is macerated for 24 hours in a quart of water; this is then boiled down to one pint and given in three fractional doses at intervals of one hour; three hours after the last dose castor oil is administered. As home medicines are recommended, pumpkin seeds (25-50) or turpentine (2-4 gm. with the yellow of an egg, repeated on several consecutive days).

Prophylactic measures are the prevention of any chance for the dogs to feed on infected organs (with regard to echinococcosis Snapper deems it necessary to forbid by law the sale of offal from slaughterhouses) and proper cleanliness of the coat to destroy fleas and lice.

(e) Tapeworms in Cats.

Etiology. Among the tapeworms parasitic in the intestinal canal of cats, only the Tænia crassicollis possesses great clinical importance, the other forms occur occasionally but do not seem to cause disturbances in health; they are T. echinococcus, Dipylidium caninum (T. elliptica), and D. Chyzeri, Bothriocephalus latus and B. decipiens.

Taenia crassicollis, a tapeworm 15 to 60 cm. long, with a globular head with four large, prominent suckers and a strong rostellum, which carries a wreath composed of 29 to 52 hooklets. Immediately after the head follow segments as wide or wider than the former; these segments increase in size from 8 to 10 mm. long and 5 or 6 mm. wide. The ova have a diameter of 21 to 27 μ.

The tapeworms develop from Cysticercus fasciolaris, which is contained in the liver of mice or rats.

Symptoms. If these tapeworms are present in large numbers they cause chronic intestinal catarrh, with poor appetite, diarrhea, then constipation, salivation, diminution of the visual and auditory power, emaciation, finally convulsions. The disease sometimes appears in an enzootic form, particularly in years when mice are very abundant.

Treatment is the same as in dogs, but the vermifuges, etc., have to be given in one-half the doses indicated for dogs.

(f) Tapeworms in Rabbits.

Anoplocephalic taeniæ are rare in rabbits, but frequent in wild hares; they include the Ctenotaenia (Tænia) pectinata, Ct. Goezei and Ct. Leuckarti, which have double sexual openings, and according to Fiebiger also the Andrya pectinata, A. cuniculi and A. wimerosa with unilateral sexual openings. (In Railliet's opinion, the Ctenotaenia is really T. Denticulata.)

Tapeworm infection frequently appears enzootically among wild hares, rarely in such wide distribution among rabbits; its clinical symptoms are a progressive anemia with emaciation and increase in the size of the abdomen. Sometimes tapeworms get into the free peritoneal cavity of the infected animals without leaving any distinct traces of perforation of the intestines (Railliet). The diagnosis of this infection can be made from finding proglottides in the feces or from postmortem examination of the animals.

The treatment consists in a preliminary administration of castor oil (tea to tablespoon doses) followed by the following vermifuge which is to be given in gelatine—or keratin capsules: extractum fil. maris

(0.5-1.0 gm.), arecanut (0.5 to 1.0 gm.), kamala (2.0-3.0 gm.), tartar emetic (0.05-0.1 gm.).

(g) Tapeworms in Fowl.

Etiology. In barnyard fowl, in pheasants and in other wild fowls of allied genera, numerous species of tapeworms are comparatively frequent and not uncommonly cause enzootic affections with fatal cases, particularly in young animals. The larval forms are taken up by chickens, by eating domestic flies, certain bugs, butterflies, worms, rainworms, and snails; in geese and ducks by swallowing certain lower crustaceans in stagnant waters; by the turkey, guinea-hen and pheasant possibly through catching certain insects.

The following briefly enumerated tapeworms occur in the several kinds of fowl:

In **chickens:** Drepanidotænia (Choanotænia) infundibuliformis (frequent; the cysticercoid lives in the domestic fly, according to Grassi); Davainea (Tænia) cesticillus (also frequent; the cysticercus is said to live in a butterfly or in a bug); Dav. (T.) proglottina (frequent; cysticercus in species of Limax); Dav. (T.) tetragona (cysticercoid in small snails); Dav. (T.) echinobothrida; Dav. cataniana; Dav. (Hymenolepis) carioca; Dicranotænia (Amœbotænia) sphenoides (cysticercoid in worms). (Cysticercoid in angleworms.)

In **geese:** Drepanidotænia (Hymenolepis) lanceolata (very frequent; cysticercoid in small fresh-water crabs of the species Cyclops); Dr. (H.) setigera (also frequent; cysticercoid in varieties of Cyclops); Dr. (H.) gracilis (cysticercoid in small crabs of the species Candona, Cypris, Cyclops, Diaptomus); Dr. (H.) sinuosa (cysticercoid in small crustaceans of the species Gammarus, Cypris, Diaptomus); Dr. (H.) fasciata (cysticercoid in Cyclops agilis); Dr. (H.) tenuirostris (cysticercoid in small crustaceans of the species Cyclops, Gammarus, Diaptomus); Fimbriaria fasciolaris (Tænia malleus) the cysticercoid of which lives in Diaptomæ.

In **ducks:** Drepanidotænia (Hymenolepis) setigera (frequent); Dr. (Choanotænia) infundibuliformis (frequent); Dr. (Hymenolepis) lanceolata; Dr. (H.) anatina (cysticercoid in small crustaceans of the species Cypris); Dr. (H.) gracilis; Dr. (H.) sinuosa; Dicranotænia (H.) coronula (cysticercoid in crustaceans of the species Cypris); Fimbriaria fasciolaris (Tænia malleus).

In **turkeys:** Davainea cataniana; Dav. (Tænia) cesticillus; Dav. (T.) echinobothrida; Dav. Friedbergeri; Hymenolepis meleagridis and H. musculosa.

In **pigeons:** Davainea (Tænia) crassula; Dav. (T.) echinobothrida; Drepanidotænia (Choanotænia) infundibuliformis; Bertiella Delafondi (Tænia sphenocephala); Bothriocephalus columbæ.

In **pheasants:** Davainea (Tænia) Friedbergeri (frequent and usually the sole cause of tapeworm disease); Dav. (T.) echinobothrida; Dav. (T.) cataniana; Hymenolepis phasiana.

In Ostriches: Tænia struthionis and others.

Symptoms. As a rule the presence only of numerous tapeworms brings about morbid symptoms in fowl. The animals are then less lively, without appetite, or they eat, on the contrary, quite a lot of feed, but they emaciate in spite of this; later on there is diarrhea, and the animals then become completely exhausted. There are also cases when death comes on quite unexpectedly and where a postmortem examination shows the presence of numerous tapeworms; in other cases there are attacks of dizziness, epileptiform convulsions, caused by marked inflammation, obstruction or perforation of the intestine.

In a tapeworm epizootic among geese imported from Russia, Cämmerer noticed nervous symptoms aside from emaciation and diarrhea. The animals became feeble

and their movements awkward; they kept on sitting quietly from the 4th or 5th day of the disease, and permitted themselves to be caught without making any effort to escape. On the 7th day they assumed a position like penguins, with the head immobilized and directed to one point or resting upon the sternum. From time to time the animals made an effort, fell, however, upon their abdomen and then remained in this position or on their side, or they again rose by the aid of their wings into the penguin position. According to Hartenstein it was in fact a case of the so-called turning-disease of geese.

Diagnosis. Tapeworm disease can be reliably diagnosticated only by finding the proglottides in the feces, or by postmortem examination, during which, however, the detection of the presence of small, almost microscopic, tapeworms of fowls also meets with difficulties. Accordingly in cases of doubt the intestinal contents or the open intestines should be washed in water.

Treatment. The treatment consists in the administration of vermifuges, which are best given in the form of pills. The most appropriate are: Areca nut (pigeons 1 gm., chickens 2 gm., geese 4 gm., young animals 1-3 to 1-2 of those doses); this drug easily leads to symptoms of poisoning in turkeys; kamala in the same doses as the preceding vermifuge; Ellinger found kamala dangerous for geese: flores kusso (1-3 gm.); sulphate of copper (blue copper vitriol in 2% solution 10-20 drops, or a solution of 1-5 to 1,000 given as drinking water); oil of turpentine (0.25-1.0 with mucilage or oil).

In America encouraging results were obtained by the administration of potash or other alkaline drugs with the feed. For every 100 birds there is added to 4.5 liters of a mixture of wheat and oats a tablespoonful of a concentrated lye prepared from the ashes of corn cobs. The mixture is slowly boiled for 2 hours, allowed to cool, and then fed to birds that have been starved for 15 hours. In ostriches Robertson found petroleum most effective (birds 1 to 2 months old, 14 gm.; 3 months old, 28 gm.; 4 months old, 56 gm.; 5 months old, 84 gm.; 6 months old, 122 gm.; 7 to 11 months old, 140 gm.; 12 months old, 168 gm.; and adult birds, 244 gm., in one day). Chickens may be fed with pumpkin seeds.

Prophylaxis. Measures of prophylaxis are: Isolation of infested birds; destruction of the droppings, after they have been collected in barrels, by sprinkling them with lime to keep away insects which act as intermediate hosts; dusting the roosts or the resting places with lime or ashes to destroy the tapeworm embryos and to keep insects away; preventing the birds from stepping into the watering troughs or feed troughs, by proper construction of the same; regular and careful cleaning of the water and feed troughs as well as the feeding grounds; periodically changing the runways and sleeping quarters and keeping flies away from the chicken yards (see prevention of fly nuisance), and burning or burying the intestines of diseased birds. **Waterfowls should be kept away from infected water.**

To destroy the intermediate hosts, cleansing substances (lime) should be added to the water. For the prevention of tapeworms in ostriches Theiler and Robertson recommended a method similar to that for stomach-worm disease.

B. Trematodes.

(*Saugwürmer* [German].)

Trematodes are hermaphroditic single worms in the shape of a leaf or tongue, with an intestinal tract ending blind, and with organs for attachment. The latter as a rule consist of a mouth sucker and a more caudally situated abdominal sucker.

In the temperate zones trematodes are found in the intestine comparatively infrequently, and usually they live there as harmless parasites. They are found more often in warmer countries where they give rise to severe disease.

More or less importance may be attached to the following intestinal trematodes:

1. **Gastrodiscus aegyptiacus.** A spoon-shaped worm, consisting of a disc, 12-15 mm. long and 8-12 mm. wide, with strongly concave ventral surface, and a larger suction-cup at the posterior pole; also a plug-shaped head with terminal suction-cup. The eggs are 150-170 μ long, oval, similar to those of the large liver-fluke, furnished with a cover at the smallest pole. The parasite has been found in the intestines of horses, asses, mules and cattle in Egypt, in Guadeloupe, Camerun, Senegal and India. It is often harmless; but sometimes it is attached to the intestinal mucous membrane in large numbers, partly in clumps, and then leads to disease with emaciation, which may continue for months. According to Nainsouta, after a massive invasion (400 to 600 from the pylorus up to the stomachlike dilatation of the colon) a rapidly progressing enteroperitonitis and even perforation of the large colon may occur.

2. **Amphistomum (Pseudodiscus) Collinsi.** A red worm which lives in great numbers in the colon of horses in India and Tonking, and causes severe disease.

3. **Hemistomum alatum.** A dirty-white flat worm, 3-6 mm. long with constriction on the anterior part of the body where two horny processes are seen. It is frequent in the small intestine of wolves and foxes, rarely in dogs. Of like infrequency in dogs is the Hemistomum cordatum.

4. **Holostomum gracile** and **H. Sphaerocephalum** are sometimes found in the intestines of ducks.

5. **Monostomum (Notocotyle) verrucosum (attenuatum),** a reddish elongated oval worm, 5-6 mm. long, with suction cup at the anterior extremity; the eggs are long-oval, 23 μ in length, on each pole a filiform process which is 160 μ long. This parasite lives in the colon and cecum of chickens, ducks and geese.

6. **Echinostomum.** Of the fascioloid trematodes of this variety the following occur: Echinostomum perfoliatum, 3-4 mm. long, at most mm. wide, whitish transparent; behind the terminal mouth suction-cup 4 thorns arranged in two rows; the brownish yellow, long-oval eggs are 100-110 μ long, they open with a cover. This parasite is frequent

in dogs, infrequent in cats (v. Rátz). **Echinostomum trigonocephalum** is rare in dogs; its eggs are 152 μ long. **Echinostomum echinatum** is a reddish, slender worm, 4-15 mm. long, the anterior portion is neck-shaped, thinner anteriorly and furnished with thorns and terminating in a head-plate which is kidney-shaped and likewise bears thorns. The eggs are yellowish-brown, 86-96 μ long, furnished with cover, the embryo is liberated in water, it invades the internal organs of various snails (Bythinia, Paludina, Limnaeus, Planorhis) and passes through a similar evolution to that of the liver-fluke.

Geese, ducks, swans and, under some circumstances, chickens become infected by consuming the encapsulated cercariæ. Echinostomum conoideum and E. recurvatum are also found in the intestines of geese, ducks, and chickens. In this category may be placed the different species of Prosthogonimus which also belong to the fascioloid trematodes and all of which occur originally as parasites of the bursa Fabricii in many species of birds, particularly in chickens. When during the growth of the bird a physiological atrophy of this organ takes place, they wander into the rectum or frequently into the oviduct, where they produce an inflammation which is frequently followed by peritonitis. Now and then they penetrate the egg before the shell is formed and are then found in the egg after it has been laid. The following species have been found: Prosthogonimus ovatus (1 to 4 mm. long, oval, with black spots; eggs brownish-yellow, 22 to 26 microns long, elliptical), P. pellucidus, P. cuneatus, P. japonicus, P. intercalamus.

7. **Heterophyes heterophyes.** At most 2 mm. long, reddish, thinner at anterior end and furnished with a fine thorn; the brown, thick-shelled eggs are oval, 26 μ long; it is rarely found in the intestines of dogs and cats.

8. **Cephalogonymus ovatus** (Distomum ovatum) occurs in the end-intestine of chickens and geese.

9. **Clinostomum dimorphum** (Distomum commutatum) sometimes causes hemorrhagic enteritis in chickens, turkeys and pigeons.

Loossia romanica was found by Ciurea in the small intestine of the dog, Loossia parva in that of the cat, and Prohemistomum appendiculatum in the intestinal tract of dogs and cats in Rumania (source of infection are fishes).

C. Roundworms. Nemathelminthes.

(a) Ascaris in Mammals. Ascariasis.

Occurrence. Ascaris and affections produced by them are frequently seen in young animals; older animals are more rarely affected and then generally only by few worms of this type. Animals which can run around free in the barn or on the pasture are especially exposed to infection, while animals which are tied can more rarely find a chance to take up the ova of ascaris (Albrecht). Enzootics have been observed among calves of 3 to 5 months (Leibenger, Gasteiger), among young pigs, occasionally among lambs (Vallisneri), foals, also among dogs.

Frequency of Occurrence of Ascarids. In 437 horses slaughtered at the abattoir of Augsburg, Stroh found ascarids in small numbers in 116 (26.5%); three times, however, there were over 300. Young worms were found all the year round. Colts particularly were infested and frequently with a large number (30 to 377); similarly army horses. Timmke found ascarids in the feces of 64% of the horses examined. According to the compilation of Schroeder the following percentages of infestations were found in dogs and cats: In Copenhagen 20.54% (dogs and cats); in the countryside in Denmark 24% (dogs); in

Iceland 2% (dogs and cats); in South Australia 27% (dogs and cats); in Japan about 50% (dogs); in Saxony 24% (dogs); in Berlin 18.5% (dogs); in Leipzig 8% (dogs) and 9.14% (cats), and in Mecklenburg 8.4% (dogs). There were found infested, according to Blanc, 71% of 49 cats; according to Galli-Valerio, 18.5% of 162 dogs in Milan, and 66.6% of 12 cats; according to Wigdor, 26.8% of 1,000 dogs in America; according to Bouin, in southern Morocco, 73.3% of 71 dogs. Freund found an unusually large number of ascarids in the entire intestinal tract of a polar bear.

Etiology. Roundworms are long, smooth cylindrical worms, with a short head and with three lips, either smooth or armed with teeth around the mouth, two spicula of equal length and numerous papillæ around the anus, in the male.

The following worms occur in domestic mammals:

1. **Ascaris megalocephala**, white or yellowish white, rigid worm; its three mouth-lips are provided with teeth, male 15 to 28 cm. long, its posterior end conical with two skin-like small wings, directed towards the abdominal surface; the female 18 to 37 cm. long, its hind end blunt, straight; the sexual pore is at the anterior portion of the body. Ova globular or somewhat elongated oval, in the feces of the host usually yellowish-brown, not segmented or only in the first segmentation stage; diameter 90 to 100 μ, surface smooth (Fig. 48). The worm lives in the small intestine of the horse, ass, and mule.

2. **Ascaris vitulorum**, with reddish-white translucent body. Male 15 to 20 cm., female 22 to 30 cm. long, genital pore in the anterior sixth of the body. The diameter of the ova is 75 to 80 μ, the membrane appears finely wavy. The worm lives in the small intestine, exceptionally also in the abomasum of calves; it is rarely found in adult cattle (Gasteiger believes that the roundworms which he found in calves were Ascaris lumbricoides).

3. **Ascaris ovis**, yellowish-white worm, mouth-lips provided with teeth; male 7 to 10, female 7 to 12 cm. long. Very rarely found in the intestine of the sheep.

4. **Ascaris lumbricoides** (ascaris suilla), body white or pale reddish, mouth-lips provided with teeth. Male 15 to 17 cm., with ventrally curved posterior end and 70-75 papillae on the anal opening, female 20 to 25 cm. long, straight; the female genital pore is at the boundary between the first and second third, situated in a ring-shaped depression. Membrane of ovum (fig. 49) wavy, ova 66 μ long. Lives in the intestinal tract of swine and man.

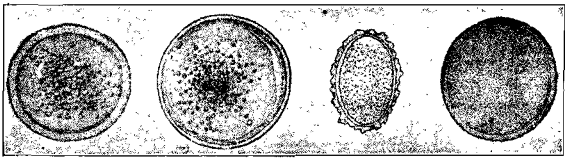


Fig. 48. Ovum of Ascaris megalocephala. Fig. 49. Ovum of Ascaris lumbricoides. Fig. 50. Ovum of Ascaris marginata.

5. **Ascaris (Belascaris) marginata**, the head of the white or pale reddish body is provided on each side with one oval wing; this makes the worm look somewhat like a lancet; male 5 to 10 cm. long; on the bent posterior portion two narrow wings, 26 papillae on each side, 5 of them back of the anus; female 9 to 12 cm. long, posterior end conical; the genital pore in the neighborhood of the first quarter. Ova globular, diameter 75 to 80 μ, membrane wavy (Fig. 50). This worm occurs

in the small intestine of the dog. (Wigdor found in America in 258 dogs infested with ascarids, Toxascaris three times as frequent as Belascaris).

6. **Ascaris (Belascaris) mystax** is so much like the preceding one that they are now held to be one and the same species; it is, however, considerably smaller than the Asc. marginata (4 to 6 or 4 to 10 cm. long; 1 to 1.5 mm. wide). It lives in the small intestine of the cat.

The **development of Ascaridae** has been demonstrated by the observations of Grassi, Lutz, Ebstein, Jammes and Martin and A. Albrecht, but particularly in experiments with Ascaris lumbricoides (suilla) on small laboratory animals by Stewart, Yoshida, and Ransom and Foster. In the presence of oxygen and a little moisture, as well as in a proper temperature, the evo-

Fig. 51. Ascaris megalocephala (one-half of natural size).

lution of the eggs, which are deposited in the ungrooved stage, begins with a rapidly progressing division and terminates with the appearance of the moving embryo. Ross found a moving embryo in eggs that had been exposed to sunlight for six weeks; Albrecht eggs capable of further development that had been kept in dry feces of a horse for six months. The time required for the embryo to develop is, under the most favorable conditions (33° C.), for Ascaris lumbricoides at least 10 days, and under ordinary outside conditions from a few to several weeks; for Ascaris megalocephala at 37° C., 3 to 4 days, at room temperature 24 to 30 days; for Ascaris vitulorum at a temperature approaching that of the body, 18 days. Only eggs containing embryos are capable of reinfecting a new host. When such

eggs are taken up by suitable animals the eggshell will burst under the exertions of the actively moving embryos. According to another conception, it is dissolved by the alkaline juices of the intestinal tract and the embryo escapes, burrows into the intestinal musoca, and in the larval stage migrates by way of the liver, lungs, trachea and pharynx down the esophagus into the stomach and again reaches the intestinal tract, whereupon, in 10 to 12 weeks after infestation, maturity is reached, and at the end of this time eggs will be found in the feces of the host. According to some reports of the occurrence of well-developed ascarids in 2 to 4 weeks old dogs (Penberthy, Marek), as well as the appearance of clinical symptoms in 2 to 3 weeks or even 10 days old calves (Gasteiger, Rozsa), some ascarids seem to complete evolution in a shorter time, if in the reported observations an intrauterine infection did not take place. In an experiment by Grassi performed upon himself, they undoubtedly reached maturity in 5 weeks.

Immigration of Ascarid Larvæ into the Intestinal Tract. According to the unequivocal results of experiments by Stewart, Yoshida, Ransom and Foster, and others, the embryo leaves the eggshell in the small intestines almost exclusively, whereupon the larvæ penetrate the intestinal mucosa and within the first two days are carried away by the blood stream through the portal vein into the liver, where they become lodged in the capillaries, causing capillary engorgement, small hemorrhages, and even ecchymosis which is later followed by necrosis in the surrounding tissues. Penetrating between the trabeculæ of the liver and along the capillaries, and, favored by rapid fatty degeneration of the liver cells, into the veins of the liver, they are then carried along the blood stream into the lungs, where they can be found on the third day following infection. After penetrating the capillaries of the lungs and reaching the alveoli, they begin their migration up the bronchi and trachea toward the larynx, pass through the pharynx, into the esophagus, down into the stomach, and finally again reach the intestinal tract, where they remain. Some of them, however, do not complete their development but are voided with the feces, as in the case of some of the embryos that did not succeed in penetrating the intestinal mucosa soon enough. The total time required for the migration of the larvæ by way of the liver and lungs and back to the intestines is, on the average, 14 days. They will be found in the lungs and respiratory tract between the seventh and tenth days following infestation. Some of them reach the lungs as early as the third day, while, on the other hand, some require a little more than 14 days to reach the intestinal tract again. During their migration the larvæ, with a buttonlike process at the front end and still contained within their cuticular egg membrane, gradually reach 5 to 10 times their original length

of 0.2 to 0.3 mm. That ascarid larvæ can occasionally enter the greater circulation by way of the capillaries of the lungs must be considered as proved by the animal experiments of Fuelleborn, in which, a few days after the beginning of the feeding experiment, ascarid larvæ in considerable number were found in the brain of the experiment animal without causing serious disturbances in the brain.

The resistance of the ascarid eggs in all stages of development is very great. They withstand intense cold and dryness without harm and live for 5 years (Davaine, Morris. Epstein, Ransom). Under unfavorable conditions, as in water or drying out, the process of evolution of unripe eggs is only retarded or temporarily brought to a standstill.

Ascarid eggs also possess great resistance toward disinfectants on account of the impermeability and insolubility of the cuticular membrane enveloping the embryo and lying under the external egg membrane. Thus, Morris succeeded in infesting animals artificially with eggs that had been kept in 2% formalin for 2 years. Furthermore, the eggs are not destroyed by 50% antiformin, which dissolves the external shell. Eukupin, sapoformal, 1 to 5% sulphuric acid, bacillol, carboxol, corrosive sublimate, 10% potassium hydroxide, and thin milk of lime also do not injure them. Phenol solutions may under certain conditions be somewhat more effective.

Natural infection occurs through the ingestion of food or water contaminated with ova, with embryos, or by licking objects where ova have been deposited. Ova voided with feces collect on the bedding straw, near watering places, on pastures, and they sometimes permanently infect the floor of certain barns: from these places they may be taken up by healthy animals and contaminate feed, water or other objects in the neighborhood of the animals (because of their manner of feeding, hogs infest themselves very easily while feeding). Railliet saw an epizootic of ascaris infection in 250 horses, due to the ingestion of infected turf straw. Gasteiger found ova of ascaris in large numbers in the manure and outside of the barn in leaf bedding, where they had been transported by the shoes of attendants and small animals (Galli-Valerio found eggs of Ascaris lumbricoides on the body of Arion and Limax snails). Sucking animals usually infect themselves, swallowing ova which have been transported from the bedding straw to the udder or by licking, in a playful manner, straw or manure infected with ova. This may explain the enzootic occurrence of ascaris infection among the calves of Miesbach-Tegernsee, where calves have a strong desire to lick soon after birth (Gasteiger). This mode of infection explains how young animals, which aside from mothers' milk receive no food or water, may become infected with ascaridae. If newborn calves suffer from diarrhea (dysentery), the ingested ova are expelled and cannot become parasitic in the intestinal tract (Gasteiger). With regard to the evolution of ascarids an intrauterine infection cannot be denied, although it is probably of a medium degree. The possibility of invasion through accidental injuries of the skin, although proved by animal experiments (hatching of eggs introduced subcutaneously and following thereupon a migration as

in feeding experiments), is hardly of practical significance. The infestivity of hog lots, barns and pastures may last for years and in the course of time even increase as the result of continuously renewed discharge of eggs in the feces of infested animals. Young animals are more susceptible to artificial and natural infestation, especially pigs up to 4 months of age. Thus, when old and young pigs are kept in the same place, all the young pigs but only about two-thirds of the old ones will become infested.

Jammes & Martin call attention to the fact that the susceptibility of the individual animals depend upon the chemical composition of the gastric juice, and this again varies according to the condition of the digestive organs, the species, age and general health of the animals. According to the authors named, acid gastric juice, as found in the stomach and possibly in the first portion of the small intestines, favors segmentation, while the alkaline intestinal juice favors the escape of the embryo out of the egg shell.

Pathogenesis. The morbid action of the migrating ascarid larvæ is most perceptible in the tissue of the lungs; on the one hand they directly injure the tissue mechanically, and on the other hand they prepare the soil in the injured lung tissue and the bronchi for a later bacterial infection. Particularly will small hemorrhages be produced following a light invasion, while echymosis of significant size will occur in massive invasions, and this is frequently followed by hepatization of whole lobes of the lungs, besides enlargement and edematous bloody imbibition of other parts of the lungs which have also assumed the color of the liver.

The roundworms exert above all a mechanical action upon the intestine, in that they irritate the intestinal mucosa by their motions, and they may occlude the lumen of the intestine partly or wholly by forming clumps; in this manner they may even cause rupture of the intestine. Even smallest scratch-wounds of the mucosa are possible, through the very fine teeth (Greim), and these may be followed by circumscribed hyperemia, inflammation, also necrosis and perforation of the intestine, in consequence of secondary bacterial infection, as well as through substances which produce local irritation and which have been shown to exist in the body-substance and in the secretions of the ascarides (Flury). The perforation occurs almost without exception at the attachment of the mesentery where there is nothing to hinder the necrotic process of the intestinal serous membrane (Francke). The ascarides are, however, not capable of directly perforating the intestinal wall.

Digestive disturbances are produced by the withdrawal of nutritive substances, also by hemolytic substances (according to Flury mainly oleic acid and acrylic acid), also by sucking the blood from the intestinal wall (Schimmelpfennig). Toxic metabolic products also are of importance, which originate in incomplete combustion and in fermentative cleavage of foodstuffs in the roundworms (Gasteiger, Flury); among these are particu-

larly volatile aldehyde of fatty acids, free fatty acids, alcohols and esters of the ethyl-, butyl- and amyl-series, also purin-bases (Flury). All these will, on the one hand, produce local irritation (following the instillation of the body fluids of Ascaris megalocephala into the conjunctival sac; Weinberg and Julien saw in two-thirds of the horses a typical reaction lasting from 12 to 24 hours with swelling of the eyelids and sometimes accompanied by dyspnea, diarrhea and diaphoresis), while on the other hand their absorption has a toxic effect on the central nervous system which may lead to clonic or tetanic convulsions, excitement, disturbances of consciousness and paralysis (Mingazzi, Greim, Flury, Dobernecker). Flury even found nitrogen-containing poisons which caused severe intestinal hemorrhage, and poisonous bases which had an action like that of atropine or conine. The volatile fatty acids which are excreted by the worms, give to the organs of the host a peculiar odor. Aside from the products of metabolism, the products of putrefaction and disintegration, of the dead worms, may also play a pathogenic rôle (Gasteiger, Flury) from which the brief aggravations of clinical manifestations, after the use of vermifuge remedies, might be explained.

Anatomical Changes. At the place of attachment of an ascaris there is formed a small round depression, sometimes covered with blood, in its neighborhood the mucosa shows catarrhal changes. In the presence of numerous worms the small intestine shows inflammatory changes along its whole extent, and deeper ulcerations may be present; perforation of the intestine occurs occasionally, and this is then followed by general peritonitis or the formation of an ichorous-purulent abscess between the folds of the mesentery, or even fatal hemorrhage (Csokor). There is, occasionally, stenosis or obstruction of the intestinal lumen by balls of worms (preferably in dogs and horses), and these may cause rupture of the intestine. Exceptionally roundworms may get into the stomach, the pancreatic or bile duct; they here produce obstruction. In exceptional cases ascarides are found, in horses, in the abdominal cavity without demonstrable injury of the walls of intestine or stomach, because the wound of perforation is already cicatrized.

Roundworms produce a peculiarly disagreeable smell in the meat of calves and lambs (Morot, Laubion, Mathis, J. Kunos, Vallisneri).

The number of roundworms is sometimes very high. A report from Brussels gave 1800 as the number of Ascaris megalocephala in a horse. Delamothe found 1215 which weighed six and a half pounds. Descamps found a mass of Ascaris vitulorum equal to fifteen liters (quarts). Krabbe 80 individuals of Ascaris mystax; Albrecht 250 individuals of Ascaris marginata in a dog six weeks old.

Symptoms. Animals exhibit morbid manifestations only in the presence of numerous roundworms, the symptoms, however, are not always proportionate to the number of worms. Most marked are the general nutritive disturbances and these are more intense, the younger the animals. The appetite is variable,

sometimes entirely suppressed, at other times much increased; in spite of this there is emaciation, the mucous membranes are pale, the coat loses its smoothness and luster. Constipation alternates with diarrhea. Migration of the roundworms into the stomach in cats and dogs causes vomiting, and occasionally one finds the worms in the vomitus, composed of mucus and bile. In horses symptoms of depraved appetite may occur, also eructations.

Symptoms of intermittent and occasionally violent colic are more frequently seen in horses, more rarely in dogs and cats. Occasionally nervous phenomena occur in horses in the shape of attacks of tetanic stiffness (Dieckerhoff, Hoffmann, Grimme), also epileptiform convulsions (Trülsen, Dubuisson), maniacal excitement followed by coma (Duncan), paretic weakness of the hind legs (Danitz), and by local sweating in the flanks. In dogs, worms sometimes cause maniacal excitement. Convulsions without any premonitory symptoms may occur during a meal, in pigs (Bru).

Rubinstein and Julien obtained in 18 out of 20 horses a positive Abderhalden reaction with the blood serum of the respective horses. Heller found slight eosinophilia (7.78%) in horses.

Sometimes the clinical picture of intestinal obturation or of peritonitis set in, or death occurs suddenly without any preceding sickness.

Sick calves and lambs emanate a peculiar, penetrating smell.

Gastsiger distinguishes two different clinical pictures in calves. In the mild form one notices a peculiar smell of the exhaled air somewhat like ether, chloroform or alcohol, the urine is light in color, there is cough, but rarely emaciation. In the severe form, aside from these same symptoms, there are disturbances of appetite, constipation or diarrhea, bloating, frequent urination, difficulty in respiration and nervous manifestations (colicky symptoms, listlessness). The duration of the disease is from one to several weeks and in severe cases a stage of convalescence which may last for several weeks.

The phenomena of ascariasis of the lungs become manifest in the first two weeks, usually about the seventh to the tenth day. They may, however, in case of continued opportunity of infestation, also appear at any time following the existing ascariasis of the intestinal tract, as is shown by the above noted observations of Gasteiger in calves and those of Raffensperger in pigs. Such disturbances are found predominantly in very young and nursing animals and manifest themselves by coughing, decreased appetite, fever, accelerated respiration and, in cases of massive infestation, by pneumonia. Light cases recover in the course of 1 to 2 weeks, while a massive invasion may terminate fatally at the end of a few days, especially in pigs (in Raffensperger's observations 40% of the pigs). Frequently a chronic emaciation with dyspnoea may follow (in Raffen-

sperger's observation in 30% of the pigs), a condition which in America is known as "thumps." This condition, however, may also be due to other forms of pneumonia in young animals.

In acute cases the migrating ascarid larvæ (see page 359) can be found in the mucus of the pharynx and bronchi. Raffensperger found large numbers of them (7 to 15 in a drop of mucus) in the bronchial mucus of pigs that died a few days after taking sick, besides young ascarids in the intestines.

Diagnosis. A reliable diagnosis can be made only after the detection of roundworms or their ova in the feces or occasionally in vomited matter. In suspected cases, vermifuges may be given to secure a diagnosis. However, experienced observers will be able to diagnosticate the disease from the peculiar smell of the exhaled air and of the urine. One may assume the presence of worms in young animals with a good deal of probability if they have previously been healthy, if digestive disturbances of an indefinite nature come on in spite of good attendance, and if in spite of good appetite emaciation develops within a few weeks. (In the few cases [13] observed at the Budapest Clinic, in which the discharged worms or those still within the intestines and the worm eggs were counted with the use of the agitation method [see page 340], figures were obtained which indicate that in dogs for every 141 eggs in 5 slides, 10 ascarids will be found in the intestines. In this respect no correlation has as yet been established in colts.)

Treatment. This varies more or less according to the species of animals. Its success should be controlled by the occasional microscopic search in the feces for ova of ascarids or by counting the worms that have ben expelled (a few eggs may still be voided [in the author's own observation in 3 dogs for 7 to 9 days] even after all the worms have been expelled).

In horses a partial elimination of the ascarids may be obtained by feeding beet roots, sugar beets, carrots and potatoes (8 to 10 liters daily with wheat bran—Moebius). According to the experiments of Hall, Wilson and Wigdor and of Hall, Smead and Wolf, which have been corroborated by the author's own observation, carbon bisulphide (carboneum sulphuratum, spiritus sulphuris) is especially effective (in about 100% of the cases all ascarids are expelled in 2 to 9 days) in doses and with a similar method of administration as for Gastrophilus larvæ. Equally good results can be obtained in colts with a mixture of different vermifuges, particularly with a mixture of filicin 6 gm., oil of turpentine 50 gm., Fowler's solution 10 gm., and tartarus stibiatus 3 gm., to 150 kg. body weight, followed by a subcutaneous injection of pilocarpin and arecolin. According to American veterinarians, oleum chenopodii anthelmintici also kills ascarids if after 16 hours of starvation 14.5 to 18 cc. (14.5 to 18 gm.) are administered in soft gelatin capsules and immedi-

ately followed by a laxative (castor oil, linseed oil or aloes). Judging from the favorable results obtained in dochmiasis in dogs, carbon tetrachloride (benzinoform, carboneum tetrachloratum) should likewise be very effective in expelling the worms (0.12 cc. or 0.2 gm. per kilogram body weight in hard gelatin capsules). Ertl obtained good results (expulsion of the worms in 3 to 4 days) with noemin (in mixture of oleum chenopodii anthelmintici, extractum granati fluidum, naphthol, sosojodolquinin, and oleum ricini, with an admixture of several kinds of fat in special gelatin capsules, to horses). Without preliminary fasting or change of diet and use of the animal, he administered one capsule in the morning and one at night, repeating the treatment 8 days later. A total of 6 to 8 capsules are given by simply dipping them in water and flipping them over the root of the tongue of the animal with the middle or index finger or with a balling gun. It is advisable to allow the animal some water after the administration of the drug. If necessary the drug may also be administered as an electuary (with sirupus althaeae and a little flour). The best of the vermifuges is tartar emetic; this drug is successful as a rule, though not always. However, this drug is not well adapted for either very young or old worked out animals, because it easily causes collapse in them; sometimes intense colicky symptoms occur after its administration, even in adult animals in good condition (Storch), especially if the drug is given dissolved in water and upon an empty stomach. For this reason Schlampp recommends the administration of tartar emetic (12-15 gm. in 1 qt. of hot water), with 1 to 1½ quarts of wheat-bran, the horse having, on the previous day, received half a ration for its noon meal, and nothing at all for the evening meal. Light horses and colts which are much affected by the treatment received, according to Lorenz' method, 30 gm. tartar emetic (dissolved in 1 qt. of water), and this divided into 6 doses, each mixed with bran for 6 successive meals. Grimme orders the tartar emetic (for grown horses and 2-4 year-old colts, 15-20 gm., for ½-1 year-olds 5-10 gm., for sucking colts 2 gm.) to be dissolved in a pailful of water and to be given three times, at intervals of an hour each, before feeding, as drinking water which has been omitted the preceding evening. According to Craig, tartar emetic is effective in the following mixture: Tartarus stibiatus 7.5 gm., acidum arsenicosum 0.3 gm., ferrum sulfuricum 7.5 gm. (daily on the feed, or in a capsule for 7 days and then followed by a laxative). Instead of arsenic (0.1-0.5 gm.) in pills or as an electuary, one may give Fowler's Solution for 5 to 8 days, morning and evening, 1 to 2 tablespoonsful for an adult horse or 1 teaspoonful for a foal, in oats, bran, or in the drinking water. Oleum terebinthinæ (50-100 gm. with 300 gm. of castor oil) has likewise been found advantageous; also arecanut (for adult horses 100-250 gm., for foals 10-50

gm., or 3 times daily, 1-2 tablespoonsful or 1 teaspoonful with moist, short cut feed, bran-mash or as an electuary). Santonin (10-25 gm. in pills or as an electuary), which, however, is quite expensive, may be used for horses, with the exception of very young foals, given in castor oil or followed by the latter.

Gasteiger had good results in **calves** with the following drugs: Tartar emetic 3-5 gm. dissolved in 125 cc. distilled water; 1 tablespoonful in milk every 3 or 4 hours, until the desired effect is brought about: areca nut with flores cinæ (mixed with the feed, calves up to 3 weeks 10 gm.: 5 gm.; up to 5 weeks 15 gm.: 7.5 gm.; over 5 weeks old 20 gm.: 10 gm.). For suckling calves Hornby found oil of turpentine 3.5 to 7 gm. reliable when administered in a gruel of linseed meal with castor oil on two consecutive days before the calf is allowed to suck in the morning (repeat after one week). A similar treatment with correspondingly smaller doses can be used in lambs, or the drugs may be administered as in the treatment of strongyles in the stomach (see page 327).

For **swine** carbon tetrachloride is administered after fasting for 24 to 36 hours and in a single dose of about 0.12 to 0.2 cc. or 0.2 to 0.35 gm. per kilogram body weight in gelatin capsules (for administration, see page 237). This drug, according to Magyary-Kossa, in the form of vermitan (with 10% of a mixture of isobornyl acetate, camphor of pine and extract of juniperus in a single dose of 0.5 gm. per kilogram body weight) expels all worms when administered in soft gelatin capsules after fasting for 24 hours and followed immediately by a dose of calomel and no water allowed till 4 hours later. According to Hall and Foster, tartarus stibiatus (0.26 gm. per 10 kg. body weight in watery solution) is also suitable; also santonin (0.13 gm. per kg. body weight) when repeated several times at intervals of 3 days or when repeated on several consecutive days (with calomel). Areca nut (for adult animals, 10 to 20 gm., for pigs, 5 to 10 gm. with honey and flour as an electuary), potassium picronitricum (0.2 to 0.5 gm. as an electuary), benzine (10 to 20 gm. with bran or with flour as an electuary), and calomel alone (0.6 to 1.2 gm.) are also recommended. According to Hall and Foster, turpentine is unreliable.

Complete success was obtained in several cases in **dogs** with a mixture of several vermifuges, particularly with one consisting of filicin 0.04 gm., santonin 0.025 gm. and thymol 0.015 gm. per kilogram body weight and followed 4 hours later by a laxative (calomel, castor oil). With santonin alone Hall and Foster obtained good results only after administering it daily for a week or longer or upon repeatedly administering it on three consecutive days with three-day intervals in daily doses of 0.013 gm. per kilogram body weight and an equal amount of calomel (also obtainable as pastilli santonini with 0.025 gm. santonin content). Thymol in a single dose of 0.13 gm. per kilogram

body weight in capsules or in a watery emulsion (after first dissolving in a little alcohol) and followed half an hour to an hour later by calomel is also effective. (Thymol is not always without danger for dogs.) Oil of thyme in the form of parasitocidin (pills containing oil of thyme, extract of yeast and yeast powder) is recommended by Neher as a good vermifuge for ascarids in dogs. In the author's own cases carbon tetrachloride (0.3 cc. or 0.5 gm. per kilogram body weight) in hard gelatin capsules was entirely satisfactory without following it later with a laxative. Hall found the combined administration of oleum chenopodii anthelmintici and chloroform very reliable (97%) when oleum chenopodii anthelmintici was given in doses of 0.3 cc. (0.3 gm.) per kilogram body weight in soft gelatin capsules and followed immediately by 0.2 cc. (0.3 gm.) chloroform per kilogram body weight in 30 cc. of castor oil, or mixed with a little castor oil in gelatin capsules and then separately the required amount of castor oil. (This treatment is not entirely without danger for emaciated dogs.) Flores cinæ (2 to 10 gm. as an infusion) has also been recommended, while Hall and Foster saw severe and even fatal intoxication following the administration of oil of turpentine. Similar vermifuges in correspondingly smaller doses may be used for cats. Previous fasting for 12 to 18 hours is necessary in dogs and cats.

Prophylaxis. Besides expelling the worms the following should be considered: Isolation of the well animals (microscopic examination of the feces); removal and destruction of the bedding and discharges of sick animals either by burning, deep burying or treating with hot lye; burning of expelled worms; frequent changes of bedding; cleaning the quarters and keeping them dry after repeated and thorough washing of the floor, posts and mangers with boiling hot water and lye to kill the worm eggs and remove the dirt by the lye (Timmke recommends vinegar instead of lye). The stalls and mangers should be thoroughly limed and the harness, curry combs, brushes, and stable tools should be placed in boiling water or vinegar or washed. Invasions of the parasites can also be kept in check by waterproofing the floors and repeatedly sprinkling them with sand and then sweeping with a sharp broom and burying the sweepings, by constructing the feed troughs in such a manner as to prevent the animals from stepping into them while feeding, and by supplying the stalls with clean drinking water. It is still better to change quarters once or twice a year (ploughing up old grounds), to keep the watering places clean, to drain off standing dirty water, and to place concrete wallows in the hog lots.

To protect the suckling pigs it is advisable, besides administering a reliable vermifuge, to cleanse thoroughly the udder and pendant parts of the body of the sows in advanced pregnancy. In large herds the dirt should be scraped and brushed

from the bodies of the sows and the udders thoroughly washed with soap and water and the sows then placed into clean quarters or box stalls, from which the fecal discharges should be regularly removed and destroyed. It is also advisable to clean the udder regularly and frequently during the sucking period.

Suckling calves should be provided with muzzles and, together with their mothers, kept away from the usual quarters of the other animals of the herd. Unless the mother animals have been successfully treated with a vermifuge, sucklings with a long nursing period (foals, calves), during which they may become infested in spite of the relatively long period of evolution of the ascaris eggs, should be treated for ascarids just before weaning. Immediately after weaning they should be placed in a clean barn, lot or pasture where infestation can not take place.

In studs and valuable herds Timmke recommends a microscopic examination of the feces of all animals for intestinal parasites twice a year. Children should not be allowed to play in places visited by hogs (the Ascaris of the pig has been found in man).

In the United States very favorable results have been obtained in combating intestinal parasites in swine with what is known as the McLean County method, which can also be used successfully against so-called filth-borne disease. According to this method the body of the sow is thoroughly cleaned and the udder thoroughly washed with soap and water 7 to 10 days before farrowing. Thereupon the sows are immediately placed in farrowing pens that have been cleaned in the manner above referred to and before cold weather sets in. These pens should be well removed from the usual hog quarters. Ten to 12 days after farrowing, or somewhat earlier, the young pigs together with the sow are removed to clean grounds, preferably to a field planted in legumes, but in all cases to such grounds (provided with movable hog houses if necessary) as have not previously been used for permanent hog pastures. In pigs that have been kept isolated from other hogs or from places frequented by other hogs from birth up to 4 months of age, an infestation from the untreated mother need hardly be feared, due to the long period of evolution of the Ascaris eggs, while the limited degree of infestation occurring in the general herd after the pigs are 4 months old will no longer be dangerous.

(b) Roundworms in Fowls. Heterakiasis.

Etiology. The roundworms which occur in fowls, belong to the genus Heterakis, which differs from the Ascarides by the presence of a suction-cup in front of the anus in the male, by the frequent inequality of the spicula and the symmetrical arrangement of the papillæ at the posterior end.

1. **Heterakis maculosa** (Heterakis columbae): body white, translucent, the head provided with three mouth-lips of equal size. The male is 16-25 mm. long, with obliquely terminating tail-end, two spicula of unequal length; the female is 20-34 mm. long, rarely as long as 70 mm., the tail-end cubical, the vulva in the middle of the body. The eggs are oval, 80-90 μ long, the egg-shell covered with a wrinkled cover of albumin. The worm lives in the intestine of pigeons, sometimes in numbers amounting to several hundreds, also rarely in the intestine of pheasants.

2. **Heterakis Perspicillum** (H. inflexa); yellow, curved worm with mouth-lips of unequal size. The male is 3-8 mm. long, with two spicula almost of equal size and terminating in a button; the tail-end terminating obliquely, and provided on each side with a membranous wing and with 10 papillae. The female is 7-12 mm.. long, the vulva in the anterior half of the body. The eggs oval. 75-80 μ long, with wrinkled cover of albumin (Fig. 52). This is a very common parasite in the small bowels of chickens, occurs frequently also in turkeys and guinea hens. Has been found at times in hens' eggs (migrating from the cloaca into the oviduct).

Fig. 52. Ovum of Heterakis inflexa.

3. **Heterakis vesicularis** (H. papillosa); a thin worm, attenuated at both ends, with two mouth-lips of equal size, and two narrow lateral membranes. The male is 7-13 mm. long with awl-shaped tail-end, two wide lateral membranes and 12 pointed papillæ on each side of the anus, spicula unequal. The female is 10-15 mm. long, vulva somewhat back of the middle of the body. Eggs 63-71 μ long, otherwise similar to those of the other Heterakides. This worm is very common in the cecum of chickens, also in that of turkeys, guinea-hens, pheasants, peacocks, geese and ducks.

4. **Heterakis dispar.** This worm differs from the preceding one by its greater length, the almost equal length of the spicula and the presence of 10 papillae laterally on the anus of the male.

Much less frequent are Heterakis compressa and H. compar (both occurring in Australian chickens), H. differens (in the posterior portions of the intestine in chickens), H. lineata (in chickens and ducks), H. isolonche, which Lucet & Henry claim not to be identical with H. vesicularis; it is responsible for verrucous typhlitis in pheasants. Ascaris crassa, which occurs in ducks, is said by Diesing to be identical with H. perspicillum.

The **evolution of the Heterakides** takes place as a whole in a similar manner as that of the Ascarides; however, with H. vesicularis (papillosa) Riley and James could not observe the migration of the larvæ by way of the liver and lungs. The eggs are voided with the droppings, and in the presence of sufficient warmth embryos develop in them (in case of Heterakis maculosa in 17 days). After such eggs are ingested with the water or feed the embryo emerges from the egg in the intestinal tract and becomes mature in about 3 weeks (observed by Unterberger of Heterakis maculosa, by Riley and James of H. vesicularis). The eggs of H. vesicularis retained their vitality in slaughtered chickens that had been kept in a frozen condition for 6 months. The resistance of the Heterakis eggs is very similar to that of Ascaris eggs.

Anatomical Changes. In addition to inflammatory changes of variable degree and extension, in pigeons infected with H. maculosa and in chickens with H. perspicillum, sometimes the intestine is obstructed by clumps of worms; in turkeys infected with H. vesicularis changes like those of ameboid typhlitis are found (v. Ratz). In pheasants infected with H. vesicularis (according to Lucet & Henry, H. isolanche) the cecum is filled with parasites; not infrequently their thickened mucous membrane is studded with pinhead- to pea-sized nodules containing mature

worms, larvæ and eggs (Typhlitis verrucosa) (Galli-Valerio; Letulle & Marotel; Klee; Lucet & Henry). In geese the circumference of the cecum is increased after a numerous invasion of H. dispar. In exceptional cases specimens of H. maculosa and of H. perspicillum are found free in the abdominal cavity without a perforation of the intestinal wall (Sabrazès & Salm; Zürn).

Symptoms. These consist in gradual emaciation, feebleness, drowsiness, diarrhea, falling out of feathers; pigeons not infrequently perish unexpectedly without having manifested any symptoms of disease. The presence of Heterakides in the intestine is shown by the demonstration of worms or eggs in the droppings; yet, in the case of rapid course, also in that of a frequent occurrence of cases, it is necessary to exclude infectious diseases because the Heterakides may be present as accidental and harmless parasites. Worm-enzootics have been produced in pigeons by H. maculosa and H. perspicillum (Railliet, Kasparek), in chickens by H. perspicillum (Rossignol, Railliet, Kasparek), in turkeys and pheasants by H. vesicularis (according to Lucet & Henry in pheasants by H. isolonche), in geese by H. dispar (Lucet).

Treatment. It is similar to that given under tapeworm infection although some of the drugs when used alone are often unreliable (Railliet's experiments with calomel, areca nut, semen cinæ and santonin were entirely unsuccessful). Most advantageous are areca nut (chickens 3.0 gm., pigeons 1.0 in pills), for pigeons also calomel (0.05 gm.); also trochisci santonini, oleum anisi or oleum rosmarini (5-10 drops in oil or as an emulsion), benzin (3-6 drops, same mode of administration); similarly oil of turpentine (2 cc. to 1-4½ kg. body weight, in the same amount of neutral oil). In the tobacco treatment recommended by Herms and Beach, 450 gm. of powdered tobacco stems are used for 100 fowls. The material is covered with water and allowed to stand 2 hours and then fed to chickens with one-half the usual ration. The chickens are previously starved for 15 hours. One hour later 312 gm. Epsom salt (for 100 fowls) is given with one-fourth the usual ration. The treatment should be repeated in one week. Although the tobacco treatment is not very reliable, it commands due consideration in cases where large numbers of birds must be treated. Those intestinal parasites inhabiting the ceca (H. vesicularis s. papillosa) can also not be expelled with certainty by other known drugs.

For chickens one might mix with their food, anis, salt, clay, also a weak infusion of garlic with ½% of salicylate of sodium (Schlampp).

Prophylaxis. This is similar to that used for the prevention

of ascariasis of mammals, especially with respect to the isolation of the well birds, burning or burying of cadavers of birds that die or of the intestines of birds slaughtered for food. The droppings should be collected and burned daily, the walls of the poultry house repeatedly scrubbed and whitewashed, the partitions scrubbed and the roosting poles, nests and other wooden parts of the poultry house planed off, the dirt floor removed and replaced with fresh sand, and the feeding and drinking vessels scrubbed at regular intervals.

(c) Whipworms in the Intestine. Oxyuriasis.

Etiology. Oxyuris are found in horses everywhere, although as a rule not frequently nor in large numbers (only Jerke observed the expulsion of 300 Oxyuris from a horse and upon post-mortem examination found 44 more).

The following Oxyuris occur in domestic animals:

1. **Oxyuris equi** (O. **curvula**), the female alone is found almost exclusively in the intestine of the horse (Fig. 53), 4 to 5 cm. long, thickened and curved in front, pointed behind, mouth surrounded by three lips; the male is difficult to find, 9 to 12 mm. long; oval ova (Fig. 54), 88 to 95 μ by 41 to 44 μ, provided on one pole with a lid-like formation. The females sometimes are up to a size of 15 cm. and their shape is then changed so that the posterior, more slender portion is 3 or 4 times as long as the anterior portion. Nitsch, Friedberger, and more recently Jerke, have claimed that this is another species which has received the name of **Oxyuris mastigodes**, while Railliet considers it as identical with O. equi and as the long-tailed variety in contrast to the short-tailed variety designated O. equi.

2. **Oxyuris vermicularis**, 2 to 3 mm. long, posterior end rolled up, female 9 to 10 mm. and straight. Ova oval, flat on one end, convex at the other. Frequently found in the rectum of man; according to Zürn also in dogs.

3. **Oxyuris ambigua** occurs in the cecum of rabbits and there produces violent inflammation. Oxyuris obvelata in guinea pigs.

Fig. 53. Oxyuris equi. Female on left, male on right.

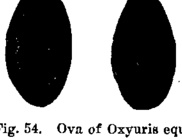


Fig. 54. Ova of Oxyuris equi.

The **development of Oxyuris** in horses is, according to Jerke, as follows: The female deposits its ova at the anus of the host, while keeping itself there, fastened with its tail. The ova adhere to the skin around the anus and of the perineum, with a sticky substance, and they develop into embryos within 1 to 3 days (eggs containing embryos retain their vitality for 2 to 4 months and at —8° C. for 6 weeks; those without embryos in a dry or moist environment for less than a month). They

then dry and drop to the floor, with detached scales of epidermis. They get out to the pastures with the manure, and may infect the feed and straw; these embryos remain alive quite long. (Roger claims that ova of Oxyuris are disseminated by flies.) After ingestion into the stomach of a host, the plug closing the ovum becomes dissolved, the embryo is set free and transported towards the large intestine, where it reaches maturity. Four months elapse between the time of infection and the appearance of mature females of Oxyuris mastigodes; of Oxyuris equi about three months.

Roger claims that Filaria irritans (see dermatitis granulosa) is nothing else but the larval stage of Oxyuris.

Symptoms. Oxyuris are found in horses, mules and asses in the large intestines and especially in the transverse colon. They move about in the intestinal contents and are never found attached to the mucous membrane. For this reason they begin to cause disturbance of health only after the female, during oviposition, has become attached with its threadlike tail to the edge of the anus or in close proximity to it and executes slight movements. This causes an acute catarrh, or more frequently an intense itching in the region of the anus; as a consequence of such irritation the animals rub their hair and the neighboring parts on hard objects and sometimes produce eczematous inflammation, which may spread along the perineum and to the thighs. The dried eggs mixed with epidermal scales and particles of dried fecal matter form yellowish-green or dirty gray adhering scabs around the anus, in the perineal region and on the lower surface of the root of the tail, under which the skin is reddened or even chafed. Hairs of the tail break off or fall out. One finds then worms in the crusts, around the anus and also on the outer surface of the feces, and on rectal exploration on the arm of the examiner after withdrawal of the former; ova are found on microscopic examination (Pflug, Roger).

Friedberger reported the case of a mare which had exhibited signs of helminthiasis for two years, and which had voided 10 to 15 Oxyuris mastigodes daily, for 9 days; Illy observed severe colic which was cured permanently after the evacuation of worms. In a case of eczema of the tail with subsequent phlegmonous abscess, an Oxyuris mastigodes was found anchored into the perineal muscles by Hahn. According to some observations, emaciation and anemia are sometimes seen. Heller observed eosinophilia of the blood (8%).

Treatment. The best treatment consists in repeated rectal injections of vinegar and soap water, possibly also solution of corrosive sublimate (1:2000). The internal administration of vermifuges is necessary only exceptionally in very obstinate cases (in Illy's case, areca nut 100 gm., followed in 4 hours by aloe 45.0 gm., had a good effect). Tartar emetic is also recommended as for ascariasis. Renaux administers 100 gm. of a 1 to 1,000 solution of corrosive sublimate in the drinking water or in a gruel. According to Railliet, thymol (15 to 20 gm. and

more) is best given with mucilaginous water and followed by a laxative. According to Hall, Wilson and Wigdor, oleum chenopodii anthelmintici (see page 364) administered in repeated doses expels all Oxyuris. In man butolan (p-benzol-phenol-carbaminicacidester) has proved most reliable. It is given in powder form or tablets (0.5 gm. each) three times a day in 0.2 to 0.5 gm. doses for 2 to 3 days and followed by a laxative. The same course of treatment is repeated. At the end of 2 weeks the whole course of treatment is again repeated (Koslowsky). Oxymors (a preparation of benzoic acid and aluminum acetate) is also effective. Two tablets are taken in water 4 times daily for 6 days. Simultaneously a lukewarm enema in which a special tablet has been dissolved is administered every night. After this, and also after each movement of the bowels, oxymors anal ointment is applied to the region of the anus (Kaufmann, Ratmer).

(d) Trichotrachelidae.

Trichina spiralis (see trichinosis) is the only one of the filiform Trichotrachelidæ which is of pathologic significance.

Trichocephali are comparatively frequent, but apparently harmless inhabitants of the intestinal tract of ruminants, hogs and dogs. Their anterior end is very fine and filamentous, the caudal end is much thicker, cylindrical, straight in the female, rolled up in the male. According to the genus, the eggs are 50-70 μ long (Fig. 55), lemon-shaped, have plug-shaped formations at both ends; in the open the embryo develops in them which matures in the intestine of the new host in the course of 2 to 4 weeks.

The following species should be mentioned:

Fig. 55. Ovum of Trichocephalus affinis.

Trichocephalus affinis, 5 to 8 cm. long; found in the cecum and small intestine of ruminants especially in sheep, goats, deer, rarely in cattle), also in the intestinal tract of hogs (Meyer).

Trichocephalus crenatus, 4 to 5 cm. long; occurs in the large intestine of the hogs and wild boar and occasionally causes intestinal catarrh, according to Haase, even a fatal enteritis or poor bodily development in young pigs, while older animals are not harmed.

Trichocephalus depressiusculus, 4 to 7 cm. long; found in the cecum of dogs, particularly of those that suffer from dochmiasis. If present in large numbers it appears to cause catarrh of the cecum, occasionally also invagination (Mégnin), at other times it may be the cause of hemorrhagic inflammation of the colon (Miller).

The Tr. echinophyllus occurs in camels and dromedaries, Tr. serratus in cats and Tr. unguiculatus in rabbits.

According to Railliet, for carnivorous animals and sheep treatments with enemas containing a suspension of thymol (see page 396) are suitable; according to Hall, repeated doses of 0.03 to 0.06 gm. santonin with the same amount of calomel given daily for one to several weeks or administered in 2-day courses with 1-day interval.

Several species of **Trichosoma** with a thin anterior portion of the body, gradually becoming thicker towards the caudal end, are found in the intestinal tract of fowl (Tr. longicolle, annulatum, collare, tenuissimum, and brevicolle); one species is found in sheep. Trichosoma columbæ sometimes produces in pigeons a severe enteritis and anemia, followed by numerous fatalities (according to Schlegel also in

homing pigeons). Freese, Schlegel and Eber observed epizootics of severe catarrhal enteritis caused by T. retusum, from which baby chicks died in 3 to 10 days and adult chickens in 2 to 4 weeks. In 92% of the cases of helminthiasis in 202 birds Eber found T. retusum most frequent, and it must be considered as one of the most dangerous parasites of young fowls. Its detection on post-mortem examination is made quite easy if a common teasing needle is slowly drawn through the mostly mucous contents of the small intestines, whereupon the exceedingly fine worms are caught across the needle and when the needle is withdrawn become visible as very fine hairlike bodies. Small flakes of suspected mucus may also be examined under the microscope. During the initial stage of the affection, oil of anise given in 5 to 10 drop doses to a tablespoonful of olive oil, proved very efficient.

(e) Dochmii in the Intestines. Dochmiasis.

(*Anchylostomiasis, Uncinariasis.*)

(a) Dochmiasis of Carnivora.

Occurrence. Dochmiasis is a severe affection, particularly of packs of hunting dogs. It has, heretofore, been observed to a large extent in France, Italy, Japan and Senegal (according to Thiroux and Teppaz, in 75% of the dogs), Punjab in British India (according to Pease, in 58% of the dogs), in America (according to Wigdor, in most of the States in 30 to 50% of the dogs) and in southern Morocco (according to Bouin, in 54.9%), but is probably also found in all other countries (reported by von Rátz, Nakoldy, Sequens and Marek in Hungary, by Jerke in Germany).

Etiology. Dochmii have the anterior end turned upward (hence the name hookworms); the chitinous mouth capsule is provided with teeth or ledges, and the male has a bell-shaped ribbed bursa at the posterior end. They really do not constitute a genus by themselves; the groups now recognized as Ancylostoma, Uncinaria and Bunostomum were formerly grouped together under the collective name Dochmius. In dogs both Ancylostoma and Uncinaria are found, and the symptoms of dochmiasis seem to be entirely or predominantly due to Ancylostoma (in one case observed by the author only a severe or fatal uncinariasis was present).

1. **Dochmius trigonocephalus** (Ancylostomum caninum, Uncinaria trigonocephalus); a whitish fine thread-worm with slightly bulging mouth-capsule; on each side of the latter there are three hooks bent inwards and attached to a chitinous plate, on the dorsal border there are two small hooks (Figs. 56, 57), not bent. The male is 9-12 mm. with spheroid, three-lipped bursa, the middle lip of which is small; the female is 15-20 mm. long, the vulva at the beginning of the last third of the body. The eggs are 74-84 μ long, 48-54 μ broad, with 2-8, rarely more groovings. The worm occurs in the intestine of dogs and cats.

2. **Dochmius stenocephalus** (Ancylostomum stenocephalum, Unc. stenocephala), somewhat smaller than the preceding one. The head end pointed, mouth capsule conical, provided with two pairs of small bent teeth on the ventral side. The eggs are oval, 63 to 76μ long, 32 to 38 μ broad similar to those

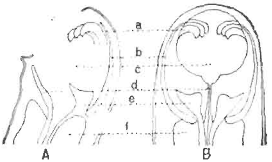


Fig. 56. Head and buccal cavity of Ancylostoma caninum (after Neumann and Meyer). A, Lateral; B, dorsal view; a, three curved teeth; b, edge of the mouth capsule; c, buccal cavity; d, stiletto-like tooth at the dorsal anterior edge; e, tooth-like body at the base of the buccal cavity; f, pharynx. (Schematic.)

of the preceding species. The parasite infests the small intestines of the dog.

The occurrence of Ancyclostoma duodenale (Dochmius duodenalis, Strongylus duodenalis Uncinaria duodenalis, Sclerostomum duodenalis) in dogs has not been proved. Young dogs can, however, be artificially infected (Looss). According to Leiper, Ancylostoma ceylonicum is found in India in many dogs and cats.

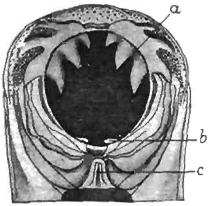


Fig. 57. Ancylostoma caninum. Cephalic end. a, Curved teeth; b, tooth-like body at the base of the buccal cavity; c, stiletto-like tooth at the base of the buccal cavity. (After Riley and Fitch.)

In man the worm causes, besides temporary gastric and intestinal disturbances, a severe anemia (ancylostomiasis, worm disease of miners, tunnel disease, brick-makers' disease, tropical or Egyptian chlorosis, anémie des mineurs; hook-worm disease). Of the same significance is, in America, Necator americanus (Uncinaria americana, Ancylostoma americanum), which is closely related to Ancylostoma.

Evolution of Dochmii. In the presence of sufficient moisture and air and in the absence of direct sunlight or in the dark and at the optimum temperatures of 25 to 35° C., an embryo hatches from the egg in 2 days; at 12° C. only after 14 days, hence under usual outside conditions in 7 to 14 days, while at 5° C. the development of the embryo is at a standstill. After hatching the rhabditiform larva completes its first molt in 3 days, thereby casting off the outside skin, and grows to two to three times its original length. The second molt follows after 8 more days, but this time the larva remains inside the loosened skin. Only these so-called encapsulated larvæ are capable of infesting the new host. They are very motile and

Fig. 58. Uncinaria criniformis. Cephalic end. a, Cutting ledges in the buccal cavity. (After Riley and Fitch.)

seek moist places (soil, water, etc.), where they attempt to climb up on objects. Under favorable outside conditions they remain viable for 6 to 7 months. After gaining access to a suitable animal the larva strips off its outside skin (third molt) at the point of entrance, and after 12 to 14 days undergoes a fourth molt, whereupon it becomes sexually mature. Eight days after the fourth molt copulation occurs and the female soon begins to lay eggs.

Natural infection is caused by contaminated, marshy water, mud or moist soil, by drinking such water, or by walking in infected water, mud or fecal matter. The disease is usually seen during the warmer season, but it may also make its onset in winter, as a case of the author's proves. Distemper or any debilitating disease forms, according to Lignières, a predisposing cause and prepares animals for infection. Infection with the larvæ takes place partly via the mouth with drinking water or food, whereupon, according to Miyagawa, some of them penetrate through the pharynx and some through the wall of the stomach, while those that are carried from the stomach into the intestines penetrate the intestinal wall and then migrate via the lungs, similarly as they do in the following mode of infection. Infection through the uninjured skin must be accepted as

proved by the unequivocal experiments of Looss and Cuillé (in man the regular mode of infection). The very motile larvæ which in some manner have reached the surface of the skin burrow into the skin within a few minutes. At first they penetrate into the hair follicles, cast off their external skin, and then penetrate into adjacent veins and lymph vessels. With the blood and lymph stream they are carried into the right side of the heart and thence to the capillaries of the lungs. Here they become lodged, penetrate the walls of the alveoli and migrate up the bronchi and trachea, over the epiglottis into the esophagus, and are then carried into the stomach by the act of swallowing. From here they reach the intestinal tract, where they remain (shown by Looss, Lambinet, Calmette and Breton, Cuillé, Miyagawa and others in experiments with Ancylostoma duodenale and Ancylostoma caninum in dogs by killing them, and by Leiper by cross-sectioning the trachea and esophagus at different intervals after infection). Some of the larvæ, however, also pass through the capillaries of the lungs, get into the great circulation and from here into the walls of the intestines (Sambon). Some of the larvæ, after penetrating into the skin, get into the lymph glands, where they become encapsulated after they have died, while still others migrate to and fro in the connective tissue for a long time and may remain alive for 5 years or longer (Looss).

Cuillé placed encysted larvæ of Dochmius trigonocephalus upon the shaved thorax of experimental dogs; after a few minutes the animals manifested excitement by howling and looking around towards the thorax, and rolling. These manifestations were caused by the irritation of the larvæ which had penetrated into the skin. Edema and erythema, which are first seen at the place of infection, disappear after 4 or 5 days, but on the day following the infestation the animals show an inclination to vomit, they are depressed; after 5 or 6 days they become voracious, suffer from bloody soft stools on the 10th day, and from the 12th to the 18th day ova are found in the feces. Death occurs usually after 20 to 30 days in very young animals, or in 10 days after a very numerous invasion. Larger dogs show the symptoms of progressive anemia but remain alive for a long time.

Pathogenesis. The Dochmii attach themselves by means of a mouth capsule between the folds of the intestinal mucosa, draw the mucosa into the mouth capsules and the esophagus, sink their peculiar chitinous teeth into the mucous membrane, and suck blood from the punctured blood vessels. Since they frequently change their location and excrete a substance which prevents the coagulation of the blood, thus causing a long-continued bleeding from the small injuries they have produced, they may in this manner, in case of a massive invasion, produce anemia. The strongly hemolytic toxin of the worms, which is absorbed by the intestines, also plays an important rôle in this respect. Besides the mechanical irritation of the intestinal mucosa, the numerous small injuries of the mucous membranes produced by the worms favor a secondary bacterial infection. Microorganisms attached to the surface of the migrating larvæ are also carried along, while microbes may also enter

the skin and the body through the small punctures (Malvoz and Lambinet), producing itching, pustulo-papulous urticaria (so-called Hautmaulwurf, creeping eruption). Even dermatitis may be produced by the larvæ migrating in the connective tissue or under the epidermis.

Anatomical Changes. At the point of attachment of the worms one sees smaller or larger hemorrhages, sometimes, also, hemorrhagic intestinal contents. The mucosa of the duodenum and jejunum at first shows the picture of acute catarrh, later on that of chronic inflammation, the intestinal wall becomes less elastic, the lumen may occasionally be narrowed. In animals just dead, one finds the worms, usually both species, simultaneously, attached to the wall; later on they become mixed with the feces; in severe cases one often sees several hundred parasites. The mesenteric and peribronchial lymph glands are enlarged, according to Lafon and Martin, all of the lymph glands being thus affected. In all fatal cases severe anemia is found, which is frequently accompanied by hydremia.

Symptoms. There are at first digestive disturbances of an indefinite nature, then anemia stands in the foreground of the clinical picture. Debility and emaciation become more and more marked, and hunting dogs become tired very easily. The skin becomes dry, the coat bristly, the extremities show edematous swelling, at first only temporary, later permanently, and which may even be followed by ulcers. At the beginning constipation is observed, later diarrhea with a periodical discharge of bloody feces, which may, however, at the beginning and also in light cases show a periodical admixture of blood streaks or at least give a positive hemoglobin test.

Examination of the blood shows red blood corpuscles, varying in size and containing irregular forms; their nnmber may be decreased so that the proportion of red to white is finally as 3:1. These findings do not, however, justify, in the opinion of the authors, a classification of this disease as pernicious anemia, as has generally been done.

According to the claims of Lafon & Martin, all of the lymph-glands become enlarged early in the course of the disease (before any other symptons are noticeable) in consequence of the invasion of staphylococci, which are, however, also found in the spleen, the blood and the intestinal contents of animals dead from the disease. It is also claimed that these staphylococci have a hemolytic effect. Another constant symptom is albuminuria (1.5 to 2%, but sometimes as much as 3-8-10% albumen in the urine), together with other symptoms of diffuse nephritis.

French and Italian authors mention epistaxis as an almost constant symptom of the disease, and the disease has received its French name from this symptom (Saignement de nez des chiens de meute). Some patients lose 100 gm. of bright red and foaming blood at one time. According to Trasbot the disease characterized by epistaxis is not identical with dochmiasis, and Péricard has recently shown piroplasmata in the blood of such patients.

Also very light cases occur in which, with or without slight intestinal and nutritional disturbances, one finds only a macroscopical admixture of blood to the feces or the presence of blood can be detected only by chemical means.

The duration of the disease may extend to one year, and all animals of a pack may become affected successively.

Diagnosis. Dochmiasis can be diagnosticated with certainty only after finding ova in the feces upon microscopic examination (in the feces, worms are found only after the administration of a vermifuge), otherwise the diagnosis can only be made with more or less probability in hunting dogs suffering from hemorrhagic diarrhea, progressive anemia, general hypertrophy of lymph glands, albuminuria and nephritis. Pentastomum tænioides likewise causes epistaxis, but profound nutritive and other disturbances and progressive anemia are absent. The abnormal condition of the feces distinguishes the disease from other forms of anemia.

Treatment. Of all drugs tried so far, Hall found carbon tetrachloride (carboneum tetrachloratum, benzinoform) to he the most effective when administered without a laxative in hard gelatin capsules in doses of 0.3 cc. or 0.5 gm. per kilogram body weight. (Whenever possible the pure drug free from phosgene and carbon bisulphide should be used.) Similar results were obtained with a mixture of carbon tetrachloride and thymol (1 cc. and 0.05 gm. respectively), or a mixture of carbon tetrachloride and oleum chenopodii anthelmintici (3 cc. and 1 cc.), when in either case the mixture was administered in hard gelatin capsules and in doses of 0.3 cc. per kilogram body weight (should not be given without a gelatin capsule or in smaller doses). Furthermore, in 85.5% of the cases Hall found effective the combined administration of oleum chenopodii anthelmintici and chloroform, when 0.1 cc. oil of chenopodium per kilogram body weight (probably also up to 5 kg. body weight 0.3 cc., for 5 to 10 kg. 0.6 cc., for 10 to 15 kg. 0.9 cc., for 15 to 20 kg. 1.2 cc.) was given in a soft gelatin capsule, and this immediately followed by 0.2 cc. chloroform (per kilogram body weight) dissolved in enough castor oil (up to 30 cc.) to produce laxation. The chloroform may also be administered in gelatin capsules and then followed by the necessary dose of castor oil (in very weak animals not without danger). On the other hand, chloroform alone was reliable in only 50% of the cases, while in 91% a complete cure was effected when oleum chenopodii anthelmintici was administered in 3 doses (0.1 cc. per kilogram body weight in soft gelatin capsules), and each dose followed by a dose of castor oil at intervals of 1 hour, and this in turn followed by chloroform in castor oil 1½ hours later. Chloroform may also be used with good results as Hermann's mixture (for a large dog, extractum filicis 4 gm., chloroform 3 gm., castor oil 40 gm.; also in the so-called white mixture (oil

of eucalyptus 2 gm., chloroform 3 gm., castor oil 40 gm.). In the author's cases the Dochmii were expelled by distol (0.04 to 0.06 gm. per kilogram body weight in gelatin capsules on 2 consecutive days). Other recommended remedies are kamala (3 to 8 gm.), with which Sequens achieved a complete cure in 1 case, and to which Mégnin recommends an addition of 0.3 to 0.5 gm. calomel and 0.005 to 0.01 gm. arsenic; extractum filicis (3 to 8 gm.) and arsenic (0.005 to 0.01 gm.); thymol (according to Sonsino, for large dogs 6 gm. in 3 doses at 2-hour intervals on an empty stomach, and 5 to 6 hours after the administration of the last dose, a laxative, if necessary. In case of necessity the treatment may be repeated once or twice at intervals of 8 days).

The patients must be nourished abundantly with milk, meat and eggs; iron preparations are indicated to counteract the anemia.

Prophylaxis. This consists in scrupulous cleanliness. Kennel and food vessels must be cleansed daily, so that the ova cannot get into the food or drinking water. The latter must be fresh and supplied so abundantly that the animals will not be tempted to drink dirty or contaminated water. Sick dogs must be separated from the healthy ones and the feces of the former must be destroyed. Dogs returning from the chase should be immediately cleaned by washing off adhering soil and mud.

Dochmiasis in Silver Foxes. According to Allen and Riley and Fitch, dochmiasis occurs quite frequently in silver foxes, massive invasions producing emaciation, retarded development and poor quality of fur. For the expulsion of the worms Riley and Fitch found oleum chenopodii anthelmintici most suitable in doses of 0.06 to 0.18 cc. per kilogram body weight (less for weak and emaciated animals), in gelatin capsules, which, when disguised in a piece of meat, are readily swallowed by the animals. One to 2 hours before treatment a small amount of food is allowed, and 4 hours after the administration of the vermifuge a liberal amount of raw liver is given, which has a very good laxative effect. Thymol (0.13 gm. per kilogram body weight in pills 2 times at 2-hour intervals) is effective in only 88% of the cases; on the other hand, however, it causes death in 18% of the animals. In small doses it was unreliable, as was also chloroform, and with the latter drug 50% of the cases terminated fatally.

Dochmiasis in Cats and Feline Beasts of Prey. Dochmiasis is but seldom observed in cats, only one enzootic having been observed in Italy by Parona and Grassi. Ancylostoma caninum (Dochmius balsami) is considered the cause; in India also Ancylostoma ceylonicum. In the zoological garden at Munich, Kitt observed 2 fatal cases in tigers, presumably pro-

duced by the new species, Uncinaria perrodens. Cases of dochmiasis in feline beasts of prey have been described by Leisering, Gervais, Diesing and others, and according to Kitt, Dochmii are found in almost two-thirds of the captured lions and tigers in the zoological gardens at Amsterdam.

In Kitt's cases the anatomical changes varied from those found in dochmiasis in dogs, in that slate-colored nodules resembling diverticula, up to the size of a bean, and containing Dochmii and their eggs, were found in the wall of the intestinal

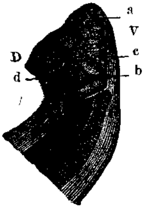


Fig. 59. Cephalic end of Bunostomum radiatum. D, Dorsal side; V, ventral side; a, edge of the mouth capsule; b, dorsal cone-shaped tooth; c and d, lateral pair of teeth.

Fig. 60. Bunostomum radiatum. Natural size. Left male, right female.

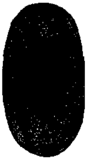


Fig. 61. Egg of Bunostomum radiatum.

tract. These nodules communicated with the lumen of the intestines by an ulcerlike opening. Some of the nodules had perforated the intestinal wall and allowed the escape of a few worms into the abdominal cavity, as a result of which a serohemorrhagic and ichorous peritonitis was produced.

The symptoms in cats are similar to those observed in severe cases of dogs (Tifo dei gatti, Italian), only their development is slower, so that after a few months pronounced disturbances and even epileptiform convulsions are noticed (Galli-Valerio). On the other hand, even manifestly diseased animals will occasionally recover (Neumann). In tigers Kitt observed an increase in the abdominal circumference as the result of ascites and peritonitis, with depressed appetite, and finally total lack of appetite, lying around and exhaustion.

Treatment is similar to that in dochmiasis in dogs, with correspondingly smaller doses for cats.

(β) Dochmiasis (Bunostomiasis) of Cattle.

Occurrence. Dochmiasis is seldom observed in cattle, especially as far as European countries are concerned, while in many regions of North America it occurs more frequently and

enzootically. Thus, according to Dawson, it occurs in southern Texas and especially in Florida, where it is known under the name of "salt sick." Also Barnett observed it in America. *(According to Stiles, 50% of the cattle in America are infested.)* In Europe it has thus far been observed only in stabled animals, especially in Bohemia (Duschanek), in Salzburg and Moravia (Reisinger), in Hungary (Marek) and in Servia (Kohn). Only animals from 3 to 12 months old show severe symptoms, while older animals show only slight disturbance of health or do not sicken at all, but may remain carriers nevertheless. (Kohn observed in a work ox pronounced anemia, emaciation and a fatal termination.)

Etiology. Dochmiasis in cattle is caused by Bunostomum radiatum (Dochmius radiatus, Ancylostomum radiatum, Strongylus radiatus, Uncinaria radiata), which is closely related to Ancylostoma.

Bunostomum radiatum (Fig. 59); 1.5 to 2.8 cm. long, head bent hooklike toward the dorsal side, mouth capsules strongly developed at the base, one well-developed dorsal tooth and ventrally 4 lanceolate teeth. In the female the vulva is situated anteriorly to the middle of the body. In the smaller male (1.5 to 1.8 cm.) the bursa is shaped irregularly and is visible to the naked eye, as are also the two spicules which project from it up to 4 mm. (Fig. 60). Eggs on the average 80 microns long, 50 microns wide, with 2 to 8 cells (Fig. 61), in freshly passed feces.

The **evolution** of cattle Dochmius is probably very similar to that of Ancylostoma. In the author's experiments with cultures (feces from cattle mixed with water and animal charcoal at 25° to 28° C.) the rhabditiform embryos emerged from the eggshell in 24 hours, and in 24 hours longer the loosened external skin could be seen on some of the larvæ. According to Stiles, the larvæ shed their skin a number of times in water or in moist material and become capable of infesting a new host in 4 to 6 weeks. The resistance of the larvæ is probably similar to that of Ancylostoma larvæ.

Natural infestation takes place by means of the feed and drinking water, manure and soil containing manure; in America, according to Dawson, particularly upon wet, marshy pastures; in Europe in the barns, if the animals are watered out of ponds, small lakes, etc. (Duschanek) and kept in so-called open stalls with adjoining paddocks and pit stalls (Reisinger) or in exercise paddocks with permanent bedding (Marek). As predisposing causes there may be mentioned poor pasturage, unfavorable weather conditions, difficult parturition, early weaning of calves, digestive disturbances and insufficient feed.

The disease is hardly ever brought into a herd by manifestly sick animals, but by carriers harboring only a few Dochmii (Reisinger). In the author's observation the disease spread to

calves from work oxen which were watered at the same trough as the calves.

Pathogenesis. The larvæ, gaining access to the body similarly to the larvæ of Ancylostoma, via the mouth or by penetrating the skin, finally localize upon the mucosa of the duodenum, or only seldom and then only sparingly, upon the mucosa of the jejunum. By sucking blood, by causing long-continued hemorrhage from the smallest injury to the mucosa and by the excretion of the hemolytic toxins they produce disturbances similar to those caused by Ancylostoma (see page 378). They may also occasionally favor secondary bacterial infection.

Anatomical changes. There is severe anemia of all organs (as in fatal hemorrhage) and hydremia with a gelatinous character of the subcutaneous connective tissue, the intramuscular tissue, the mesentery and the heart wall. The contents of the duodenum and sometimes also of the jejunum are found more or less mixed with blood, or the contents may be of a thin consistency and blood can be detected in them only on chemical examination. The mucosa shows slight swelling and has partly a slate-colored, partly a slightly reddened appearance, besides presenting numerous hemorrhages up to the size of a pinhead. Dochmii will be found partly free in the lumen of the intestines, partly adhering to the mucosa. The number of worms may vary considerably; Reisinger counted as many as 1,000 specimens; in one case 4,219 were counted (Csontos and Pataki), and in another undoubtedly still more.

Symptoms. Not infrequently the first symptoms are a morbid desire to lick, and to lick certain parts of the body (especially the feet) until they become sore, probably as the result of irritation at such points produced by the penetration of larvæ through the skin (Reisinger). These symptoms may be absent in all animals of an infected herd (own observations) or appear only in the further course of the disease. In such cases the disease begins a short time after infection, with a rapidly progressing severe primary anemia and retarded development, while for the time being emaciation is relatively less pronounced and the appetite but little depressed. Colicky symptoms also appear. Following attacks of colic or without them, diarrhea often develops and then frequently alternates with constipation and a medium degree of bloating. Edema in the region of the throat, on the extremities and in the body cavities is observed relatively early. Pronounced exhaustion develops rapidly and the animals finally die in a cachetic condition.

The course and duration vary considerably according to the degree of invasion and the age of the animal. Aside from those cases harboring only a few worms and which do not show symptoms of the disease, cases are observed with a duration of from

2 weeks or a little longer up to several months. Furthermore, cases often occur which are permanently light in character and heal spontaneously, following which the animals remain carriers at least for a time. Such cases are especially frequent. (Reisinger observed in cows soon after being placed in an infected calf barn a falling off in milk flow and condition, but no other symptoms.)

Diagnosis. A correct diagnosis depends upon the microscopic detection of the characteristic ova in the feces. By the use of the agitation method (see page 340) and by counting the eggs in 10 preparations of fecal suspension, the degree of infestation can be roughly estimated, since, according to the investigations of Csontos and Pataki, a total of 5 eggs in 10 preparations corresponds on the average to 100 sexually mature Dochmii in the intestinal tract. Likewise dochmiasis can also be distinguished from gastrointestinal strongylosis only by microscopic examination of intestinal discharges. The eggs of the strongyles (see page 324) are much larger and contain embryos or a larger number of cells. On the other hand, it is difficult to exclude œsophogostomiasis, even by a microscopic examination of the feces, since the eggs of the two resemble each other very closely. With a little practice a decision is possible, however, particularly by taking into consideration the fact that the eggs of Dochmii are usually shorter and hence appear plumper and contain only 2 to 8 cells, in contrast to the eggs of Œsophogostoma, which contain 16 to 32 cells (compare Fig. 61 and Figs. 65 to 67). In specimens of feces already 2 to 3 days old this difference is no longer so apparent, due to the rapid division in the eggs of Dochmii. Enteritis paratuberculosis differs from dochmiasis by a usually continued diarrhea with watery stools in which, and in scrapings from the anterior rectal mucosa, acid-fast bacteria are found in large numbers.

Treatment. Distol has been entirely satisfactory in 63 cases in yearlings when administered at least twice, or, better, three to four times in succession at intervals of from one-half to 1 day. (For doses and method of administration see chapter on liver-fluke disease). The blood-sucking Dochmii have undoubtedly become fatally intoxicated by taking up the active principle of filicis (male fern), which is rapidly absorbed even by the stomach, and hence the worms appeared in the feces in large numbers. Following such treatment there was ascertained in a closely observed animal an increase in the number of red blood corpuscles from 3 million to 8 million, the hemoglobin content rose from 25° to 50°, according to Sahli, and the body weight increased from 135 to 190 kg. Duschanek successfully used kamala with aloes (āā 5 gm. daily till diarrhea became manifest). Gattinger used kamala alone less successfully (1 dose of

15 gm. weekly), also flores sinæ (1 dose of 50 gm. weekly). According to Reisinger, the repeated administration of oleum terebinthinæ (2 to 3 tablespoonfuls in olive oil) is also successful. The favorable result of the treatment is much enhanced by placing the animal in a barn where the feces and litter can be removed daily, also by pasturing the animal. Such precautions may, even without medical treatment, bring about a decrease in the enzootic or produce spontaneous cure in light cases.

Prophylaxis. The following prophylactic measures are recommended: Sanitary barns, especially daily removal of manure and exclusion of permanent bedding; exercise stalls and pit stalls; provision for clean drinking water, if possible running water or well or spring water; burning or burying manure from sick animals and from carriers, or at least mixing the manure with hydrated lime or sprinkling it with milk of lime if it cannot otherwise be placed in a safe place. In case of infected pastures a change of pasture is indicated (the pasture may be used by other species of animals).

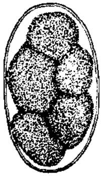


Fig. 62. Egg of Bunostomum cernuum.

Dochmiasis in Sheep and Other Animals. According to Jerke, Bunostomum cernuum (B. trigonocephalum, Ancylostoma cernuum, Dochmius cernuus, Monodontus trigonocephalus) is a common intestinal parasite of sheep which, as a rule, is found only in small numbers and therefore does not cause any symptoms of disease. Occasional massive invasions, however, produce a clinical picture resembling in detail dochmiasis in cattle and also produce similar anatomical changes. Less frequently they are found in goats. (It remains to be investigated whether the enzootic anemia reported by Walker and Baldrey in sheep and goats in Punjab (British India), which presents a clinical and anatomical picture similar to severe cases of dochmiasis, is not likewise caused by Dochmii, for these are frequently observed in dogs in that country.) Ströse saw digestive disturbances caused in a hog by Dochmius (Globocephalus) longemucronatus (Characostomum longenum). What Ráthonyi described as dochmiasis in nine horses was a strongylosis, as shown by v. Ratz. Schmaltz found Uncinaria os papillatum (Bathmos-

tomum Sangeri) in two elephants with hemorrhagic enteritis anemia and hydremia.

(f) Palisade Worms in Horses.

Sclerostomiasis equorum.

Etiology. The Sclerostoma (strongyles) which occur ver frequently in the intestines of horses, have an obtund anterio end, which is straight or hent ventrally; the mouth-opening i circular, the mouth-capsule is chitinous, provided with palisade like teeth (lip fringes), and with a dorsal longitudinal ray.

1. **Sclerostomum equinum,** Müller (Strongylus armatus, Rudolphi; Str. ne glectus Poeppel, Scl. quadridentatum, Sticker). The male is on an average 35 mm long, 1.25 mm. thick; the female 45 to 47 mm. long and 2.25 mm. thick. Mout capsule almost regularly long-ellipsoid, with 4 high narrow teeth below them, th dorsal ones densely crowded, the ventral ones separated. The bursa at the tail en of the male wider than long, the median flap developed to a lesser degree. Th genital pore of the female about 14 mm. in front of the caudal end. Ova oval, thi shelled, 65 to 80 μ long by 45 to 50 μ wide. Larvæ 0.58 to 0.88 mm. long, simila to those of Scl. edentatum only shorter; its anterior end somewhat conically pointe (Albrecht). This sclerostoma is found least frequently in the cecum and colon

2. **Sclerostomum vulgare,** Looss (Strong. arm., Rudolph; Scl. bidentatum Sticker) claimed to be a larval form of the preceding one by Rudolph; howeve really a new species, the larvæ of which are concerned in the production of so called worm aneurysm (see page 256). Male at the utmost 15 or 16 mm. long an fairly uniformly 0.7 mm. thick; female 23 or 24 mm. long and 1 mm. thic slender towards the tail end, mouth capsule like a shallow goblet. Two teeth at th lower end of the dorsal depression of the mouth capsule; these protrude as ear-lik projections. Bursa of the male with three flaps, the median one, larger than tha of the preceding species, is overlapped by the lateral flaps. The female genital por 8 mm. anterior to the caudal end. Ova 70 to 80 μ long and 43 to 52 μ wide. Th anterior end of the larvæ (0.7 mm. long) is somewhat rounded off, the posterio portion gradually diminished into a filiform tail end, which is about one-hal shorter than the body. Around the gut 32 larger cells are usually arranged i double rows in mosaic form; the sexual apparatus is situated on a level with the 13t intestinal cell (Albrecht). It is frequent in the cecum and colon; the larvæ ar often concerned in the formation of so-called worm-aneurisms.

3. **Sclerostomum edentatum,** Looss (Scl. edentatum, Sticker). Length of male 23 to 25 mm., greatest thickness about 1.5 mm.; length of female 33 to 36 mm., thickness about 2 mm. Head globular, distinct from body; mouth capsule goblet-like, teeth-like formations totally absent; however, at the place where the ventral teeth are situated in Scl. equinum, there are some small nodular elevations. Bursa of the male very similar to that of Scl. equinum. Genital pore of female 9 to 10 mm. anterior to the caudal end. Ova (Fig. 63) 65 to 88 μ long and 45 to 50 μ wide. Larvæ 0.55 to 0.88 mm. long, body almost one-third longer than the gradually tapering caudal end; marking of the intestinal cells indistinct, thickness of the body less than that of larvæ of Scl. vulgare (Albrecht). It is very frequent in the cecum and colon of horses, sometimes isolated, sometimes in large numbers. According to Martin, it was found in 42.2% of 426 horses examined; colts up to 2-3 years are affected more frequently.

Fig. 63. Ovum of Sclerostomum edentatum.

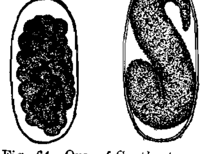


Fig. 64. Ova of Cyathostomum tetracanthum. On the right side ovum with embryo from horse manure preserved one day.

4. **Cyathostomum tetracanthum** (Strongylus tetracanthus Mehlis, Sclerostomum tetracanthum), white worm; male about 9 mm., female 10 to 12 mm. long. Mouth capsule fairly shallow, narrowed in the middle, fine triangular leaflets, arranged in a radial manner and looking from above like a wreath of fine bristles (called by Looss internal leaf wreath) spring from the narrowed place of the mouth capsule. In front of the anterior margin of the mouth capsule there is an outward projecting wall formed by the skin, this wall is likewise provided with radially arranged leaflets (external wreath of leaves). Sexual pore at the posterior end, directly in front of the anus; ova (fig. 64) oval 90 to 110 μ long, 40 to 50 μ wide. The caudal end of the larvæ (0.46 to 0.80 mm.) is about one-third longer than the body proper, anterior end of the latter somewhat more pointed, the union of the body proper and the tail end well marked; the intestinal tract only shows 8 or 9 larger alternating cells, on a level with the fifth of these cells the sexual apparatus is situated (Albrecht).

As the representative of a third genus Looss distinguishes **Triodontus** which has an almost globular small but thick-walled mouth capsule, which possesses no real teeth, but the chitinous lining of the three-cornered lumen forms three projecting tooth-like formations. The genital pore of the female is situated near the caudal end. Two species belong to this genus: Triodontus minor and Triodontus serratus. They do not, however, appear to occur in Europe. It is also frequent in the colon of horses (Fiebiger found it in about 25% of the examined horses, Kotlán in practically all horses examined in Hungary). Many other species of Cylicostomum are also found.

Representatives of the following species are found less frequently: Triodontophorus (Kotlán found it in 60% of the 30 horses examined), Gyalocephalus (according to Kotlán, quite frequently), Poteriostomum, Craterostomum, Oesophagodontus.

According to Leuckart, Cobbold, Sticker, Martin, Barthel, A. Albrecht and others, the **development** of Sclerostomum and of Cyathostomum tetracanthum is as follows: In the ova, which are voided with the feces of the horse, embryos develop in the outside world, under favorable conditions of temperature, in 3 to 4 days, later in winter, which after hatching, become larvæ 0.4 to 0.8 mm. long, round with filiform, caudal ends. In water or in dry dust, the larvæ can live only a few days, but they may remain alive for months in moist or at least not too dry horse manure. After 15 to 20 days, or at a temperature of 30° C., shedding takes place in a few days; the detached cuticle is completely shed, however, only after several months.

With the beginning of the molting the resistance of the larvæ is increased to such an extent that they can now live in water for 6 to 8 months and withstand a temperature of 0° C. without harm. According to experiments now in progress, Sclerostomum of horses seem to produce a free-living sexual generation.

Opinions differ as to the further development of the larvæ in the body of the horse. The majority of the authors (Leuckart, Railliet, Sticker, Barthel and others) believe that the further development of the larvæ to sexual maturity depends upon their passage through the circulatory system and spending some time in the blood vessels. The larvæ are said to penetrate the intestinal mucosa, thus gaining access to the portal vein, and are carried through the liver, in the capillaries of which some of them are retained; the others pass on to the right heart and then to the lungs, in the capillaries of which some are likewise retained. Those larvæ that are retained in the capillaries of the liver and lungs later perish. The larvæ of Sclerostomum vulgare (bidentatum) which pass into the great circulatory system reach the mesenteric artery, where they become lodged in the vasa vasorum of the media (Schuetz, Sticker) and produce an inflammation which is later followed by an inflammation of the intima. According to others, they attach themselves directly to the intima (see page 257). The larvæ probably also reach the

right heart through the lymph stream, thus passing around the liver. Others express the opinion that the larvæ, after penetrating the intestinal mucosa, actively migrate to the mesenteric artery and there penetrate the wall of the blood vessel. In contrast to this, others (Colin, Willach, Olt, Adelmann) believe that the usual evolution of the larvæ consists in encapsulation in the wall of the intestines and that those reaching the circulatory system are only stray larvæ. Schlegel and Adelmann also believe that the evolution can also take place directly in the lumen of the intestines without penetration of the wall. (The absence of immature worms in the lumen of the intestines does not bear out this belief.)

The migration of the larvæ of Sclerostomum vulgare (bidentatum) from the blood vessels into the intestines takes place possibly by their being carried along with the blood stream after the larvæ have ruptured their external skin and have again become free. They now become lodged in the small arteries of the large intestines, produce worm nodules and grow to the length and form of the sexually mature worms. In the worm nodules they probably undergo two more molts and finally escape through a craterlike opening into the lumen of the intestines, where they become mature. According to Glage, the period of development is 1 year; therefore sexually mature Sclerostomum parasites are not found in colts under 5 months old, in spite of the fact that the mesenteric arteries may be packed with larvæ.

The main place of evolution of the larvæ of Sclerostomum edentatum is in the subserosa of the peritoneum (especially under the parietal layer), to which place the larvæ are probably carried by the blood stream. Here they go through three molts, and after about 3 months actively migrate to the intestines, whereby they strive to reach the mesenteric artery. They migrate down between the two layers of the peritoneum along the adipose tissue surrounding the blood vessels and finally penetrate the wall of the intestine, producing worm nodules similar to those caused by larvæ of Sclerostomum bidentatum. They escape from these nodules into the lumen of the intestines and become sexually mature.

The larvæ of the genus Cyathostomum s. Cylicostomum are said to penetrate only into the mucosa of the large intestines and there produce worm nodules.

Nothing definite is known about the further evolution of the larvæ of Sclerostomum equinum. It must, therefore, remain doubtful whether their evolution to sexual maturity takes place directly in the lumen of the intestines of the new host or whether the larvæ, like those of Cylicostomum, probably also migrate into the body of the horse at least to some extent.

The **invasion** with larvæ is brought about by the consumption of contaminated water, by the consumption of feed and bed-

ding to which fecal matter may adhere, or by licking moist walls of the barn; also by grazing on marshy pastures, by the consumption of hay from swampy meadows, and, according to French veterinarians, by the consumption of alfalfa and clover. In horses at pasture the greatest number of Sclerostomum probably develop in late spring, summer and early fall.

Pathogenesis. All representatives of Sclerostomum, also the species of Cylicostomum found free in the lumen of the intestines at postmortem, appear to suck tissue juices and blood (especially the Cylicostomum). They also produce small erosions at the point of attachment, which favor secondary bacterial invasion, similarly as do the injuries produced by the penetration of the larvæ and the microbes carried along with them. Furthermore, a strongly hemolytic toxin has been found in mature Sclerostomum (Bondouy). In spite of this, disturbances in the health of the animals become manifest only after massive invasion.

Anatomical Changes. The number of sexually mature Sclerostomum that may be found in the large intestines varies from a few specimen to hundreds, thousands, and exceptionally millions (Scherenberg). Thus, Ludewig counted 80 Sclerostomum vulgare and 800 to 1,000 Cylicostomum tetracanthum in a portion (15 cm. long) of the apex of the cecum of a horse, while Eber found thousands of Cylicostomum in one case. In the still warm cadaver Sclerostomum equinum, S. vulgare, S. edentatum and S. triodontophorus are found attached to the mucosa, and upon a forcible removal one may see a small buttonlike prominence consisting of proliferated submucous connective tissue. The smaller Cylicostomum are found as actively moving white or red worms in innumerable numbers in the intestinal contents between the particles of hay or straw, from which they can be distinguished only upon strong dilution of the contents in a glass dish. The worm nodules in the submucosa produced by the larvæ (sclerostomiasis, s. strongylosis nodularis intestini), especially in the cecum and here again predominantly at the apex, less frequently in the large colon, seldom in the posterior end of ileum or the first part of the small colon, may also be only few in number or placed close together. (In Ludewig's case 3 to 4 worm nodules, probably produced by larvæ of Cylicostomum tetracanthum, per square centimeter were found in the mucosa of the apex of the cecum; in another case reported by Eichhorn, 20 worm nodules of similar origin were found per square centimeter.) According to the age and often the genus of the enclosed larvæ, the nodules sometimes resemble intestinal follicles, vary in size up to that of a millet seed, are flattened, dark gray to reddish, with a slitlike opening that can be detected with a microscope, and contain a coiled, reddish larva (mostly Cylicostomum tetracanthum). On the other hand, the

nodules below the mucosa frequently reach the size of a pinhead, pea or bean, very seldom the size of a walnut, are soft in the center with a turbid, reddish-yellow, bloody, purulent content, an indurated wall, a hardly visible opening, and also contain coiled larvæ, mostly of the other species. Not infrequently the larvæ are no longer found in the nodules, but in this case an ulcer or a craterlike opening leading into the lumen of the intestine will be found, which may be mistaken for an ulcer as found in glanders. The ulcers or craterlike openings will later be displaced by a grayish-white, smooth scar. In case of Triodontophorus almost perforated ulcers similar to those found in hog cholera were seen (Ransom). Many of the nodules will become calcified. Following massive invasions, especially with Cylicostomum tetracanthum, there is found a more or less severe circumscribed or diffuse inflammation of the mucosa of the cecum and large colon with a bloody character of the thin, pasty or liquid intestinal content, which may even appear like black liquid blood (Scherenberg). The larvæ of Sclerostomum vulgare also produce an arteritis and thrombosis, especially of the mesenteric arteries (see page 257), and a short time after invasion sometimes also a traumatic hepatitis, as do probably also the larvæ of other species (made probable for Cylicostomum tetracanthum by the observation of Eichhorn). Sometimes abscesses containing larvæ are found in the mesenteric lymph glands. Bang observed intramuscular abscesses and on one occasion nephritis.

In acute cases of sclerostomiasis following invasion with larvæ of Sclerostomum edentatum there is found in the peritoneal cavity a purely serous, sometimes slightly cloudy exudate with flocculi of fibrin, also serous infiltration of the subperitoneal adipose tissue, and hematomes or hemorrhagic spots up to the size of a walnut or a hen's egg, particularly under the parietal layer, in the mesentery or under the intestinal serosa, not infrequently also under the fat capsule of the kidney. Each of the hematomes contains an already sexually differentiated worm, which, when situated near the surface, appears like a varicose vein. Exceptionally, a fatal peritonitis develops after the perforation of the worm nodules, or again infarction of the kidney takes place with a fatal hemorrhage into the renal capsule, or infarction of the spleen, followed by rupture of that organ. At the same time the intestinal mucosa contains worm nodules and shows circumscribed or diffuse hemorrhagic spots.

In severe cases the anatomical findings are supplemented by signs of anemia, hydremia, emaciation and cachexia.

Symptoms. Clinical symptoms in sclerostomiasis of the intestines are produced particularly by an invasion of large numbers of Cylicostomum, by the sexually mature worms as also especially by the larvæ located below the epithelium of the

mucosa, and under certain circumstances partly by other Sclerostomum and their larvæ (Sclerostomum vulgare), and last from 1½ to 2 months. Many cases are also observed in colts (Schwarzmeier, Penberthy, William, Bochberg Leneveu, Covault), especially in late fall and early winter after the colts have been placed on wet pastures, whereby colts only 4 months old may suffer with severe cases (Schlegel).

Light cases of slowly developing sclerostomiasis are characterized by poor development, loss of condition, and rough coat of hair in spite of good care and feed. Frequently also circumscribed perspiration, particularly in the region of the flank, is noticeable, and the animal easily tires while at work. Occasionally intestinal sounds, foul-smelling soft stools and periodic attacks of colic are observed. Severe cases manifest themselves by diminished appetite, or feed and water may be entirely refused; rapidly progressing emaciation, staggering gait, anemia, and acceleration of the pulse. Besides these, severe, foul-smelling diarrhea is also observed, frequently with the passage of Sclerostomum and later with edema, which may be quite extensive as in purpura hemorrhagica. Even metastatic abscesses, arthritis or a general infection accompanied by fever may be observed.

Sclerostomiasis produced by the migration of the larvæ of Sclerostomum edentatum under the peritoneum (sclerostomiasis enzootica [Schlegel]) is observed mostly in colts, and occasionally manifests itself only in gradually progressive anemia and emaciation for months or even years, or again in permanent or periodical diarrhea or periodical attacks of colic, especially after exercise; in other cases in acute peritonitis. The temperature rises rapidly to 40-41.9° C., sometimes only to 38.5-39.6° C.; the pulse up to 78-80-100 per minute; it becomes weak and finally can no longer be felt. Respiration is rapid and accompanied by groaning. There is great prostration, lack of appetite, yellowish or brownish-red discoloration of the mucosa. Occasionally there are attacks of a mild colic, urination is exceptionally frequent and the urine shows a red discoloration. The symptoms gradually become more intense and finally profound anemia and cachexia develop. Most patients die after a few days (acute form); in others the disease lasts up to seven weeks. Some animals, however, are not very seriously sick and recover, while those affected profoundly all die. Isolated hemorrhagic spots with larvæ of sclerostoma are frequently found in horses that are otherwise healthy, upon being killed in the slaughtering house.

Diagnosis. The presence of mature Sclerostomum in horses may be recognized frequently on rectal examination when some of the worms are brought out on the arm of the examiner. As a rule, however, the diagnosis is made only upon finding ova by

microscopic examination of the feces, for which the method of Adelmann and Albrecht (see page 342) may be used for the determination of the larvæ under consideration of the above-mentioned characteristics, as the presence of an eosinophilia of the blood would indicate sclerostomiasis only when other parasites can be eliminated and Sclerostomum are present in large numbers (Schermer).

Since Sclerostomum may be present in the intestine of the horse without giving rise to any disturbance in health, it is necessary, in the presence of symptoms of disease in which Sclerostoma are found, to make a careful examination of the animals and to consider all conditions of environment (wet pastures, time of taking sick, etc.) and the number of eggs found before the morbid condition can be ascribed to Sclerostomum. In this case it must also be considered that in cases in which the disturbances are exclusively or predominantly due to worm nodules or subperitoneal migrating larvæ, the absence or presence of only a small number of eggs in the feces has no diagnostic significance. Bloody or chronic diarrhea associated with emaciation in young horses in summer is always suspicious of sclerostomiasis.

The differential diagnosis must consider the socalled wasting of foals (Dieckerhoff) (see page 192), especially also infectious anemia (see Vol. I); the latter is very similar in its clinical and pathologic characteristics to sclerostomiasis as described by Schlegel.

Treatment. To combat intestinal sclerostoma one may make use of the same means which are recommended for ascariasis (see page 364), with the exception of carbon bisulphide, which is not effective (only Leneveu uses it). Hall, Wilson and Wigdor found oleum chenopodii anthelmintici very effective when administered after the animal has been starved, the drug being given in single doses of 16 to 18 cc. (16 to 18 gm.) in soft gelatin capsules and followed immediately by aloes or linseed oil (up to 1,000 cc.). Very good results are obtained with oil of turpentine (60 to 100 gm. with 500 gm. castor oil). (Popescu saw the expulsion of numerous worms after the administration of 120 gm. of oil of turpentine in 2 doses, followed 3 days later by 80 gm. castor oil.) Eichert recommends 2 doses daily of creosote, 1 gm., internally; Liénaux 1 to 3 gm. arsenic for 3 days, besides intestinal disinfectants with mucilaginous drugs which are fed to the animal with fresh milk and gruel. Atoxyl and sodium arsenate (0.5 to 2 gm. internally in increasing doses) may be tried. Eichhorn successfully combats the disease by the administration of Fowler's solution (1 teaspoon to 1 tablespoon three times daily). Schuemacher always had good success with antistrongylin (see page 364). According to Railliet, thymol in watery emulsion (see page 396) as an enema should be tried,

or as a fine powder as a bolus per os. Giles observed in a pony the expulsion of most of the Sclerostomum after the administration of three doses of 15 gm. thymol each; Theobald after two doses of thymol of 1 gm. each in 30 gm. of alcohol, followed by a laxative, by which also the larvæ in the worm nodules are said to have been killed. Since in another case of Giles's an enema with 45 gm. thymol in alcohol was fatal also to the host, Railliet warns against the use of alcoholic solutions of thymol. Intravenous or subcutaneous injections (beginning with 0.2 and increasing to 0.5 or 1 to 1.5 gm. in 1 per cent saline solution [Bochberg] or 3 gm. in water at 1 dose [Dorn]) have repeatedly been successful. All such drugs should be periodically repeated so as to kill the worms which have in the meantime escaped from the worm nodules into the lumen of the intestines. The animals should also be well fed and kept away from pastures.

Concerning prophylaxis, see page 271.

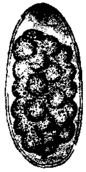


Fig. 65. Egg of Oesophagostomum radiatum.

Fig. 66. Egg of Oesophagostomum venulosum.

Fig. 67. Egg of Oesophagostomum dentatum.

(g) Oesophagostoma in the Intestines. Oesophagostomiasis intestinalis.

(*Oesophagostomiasis nodularis* [Marotel]; *Maladie nodulaire, Helminthiase nodulaire ou larvaire, Folliculite caséuse, Dochmiase larvaire* French].)

Occurrence. Diseases caused by the larvæ of Oesophagostomum occur in all parts of the world, but preferably in Europe and America, mostly sporadic but also enzootically, and then with high mortality. Cattle and sheep are affected most commonly, more rarely goats and pigs. Oesophagostomas are also occasionally found in the intestines of deer, monkeys and even in man.

Etiology. Oesophagostomas have no mouth proper because the opening representing the mouth leads directly into the oesophagus. The following oesophagostomas occur in domestic animals:

1. **Oesophagostomum radiatum** (Oe. inflatum, Ankylostomum bovis, A. radiatum, Strongylus dilatatus), a whitish worm with ring-shaped mouth-capsule, a cuticular swelling at the anterior end, behind it two broad lateral membranes. The male is 14-15 mm. long, with slight tri-lobulated bursa; the female 15-20 mm. long; the vulva is near the anus, surrounded by a ring-shaped swelling. The eggs are 70-76 μ in length, similar to those of dochmiae. The worm lives in the terminal portion of the small bowel and in the colon of cattle.

2. **Oesophagostomum venulosum.** This worm occurs in European sheep and goats.

3. **Oesophagostomum columbianum** lives in the intestines of sheep in America.

4. **Oesophagostomum dentatum.** Male 8 to 12 mm., female 12 to 25 mm. long, without cuticular enlargement and without lateral membranes at the cephalic end; eggs, similar to those of the foregoing, only a little smaller (Fig. 67).

The **development of Oesophagostomum** takes place, according to Marmotel, as follows: From the ova voiled with the feces of the animal host, embryos are at once hatched if heat and moisture are sufficient, and then undergo, according to Veglia, an evolution similar to that of Haemonchus contortus. After molting they soon become encysted and capable of infesting a new host, are then very resistant and retain their vitality in the outside world for one year. At night when the grass is moist, or on rainy or foggy days, they migrate toward the tips of blades of grass, but on bright days with sunshine they crawl back into the soil. If the larvæ are taken up during the months of August and September, they bore at once into the intestinal wall, and remain for 6 to 7 months in the submucous connective tissue, where they successively develop, first, into strongyliform, then anchylostomiform, finally œsophagostomiform larvæ. During March and April, they wander into the intestinal lumen, and here they attain sexual maturity during May and June, their full length only in July and August, then they copulate, and the females deposit their eggs. The maturation of Oesophagostomum dentatum, however, occurs within the intestinal worm nodules (Seiler); the same is also the case in the Oesophagostomum living in the intestines of man.

Corresponding to the life history of the larvæ, **invasion** takes place on wet, swampy ground in the months of August and September, and in stabled animals all the year round if the environment is suitable for the further development of the embryos and larvæ. (According to Walker, the larvæ of Œsophagostomum penetrate the skin of man similarly to the ancylostoma larvæ.)

Pathogenesis. Œsophagostomum seem to be very common in cattle and sheep and occur frequently in goats. Among several hundred cattle and sheep and a number of goats from different parts of Hungary examined by the author but few animals were found which did not have Œsophagostomum eggs in their feces. In comparison with their frequency mature worms do not seem to cause a catarrh of the large intestines even when present in large numbers. (In a goat which did not show any noticeable intestinal disturbances 369 Œsophagostomum eggs were counted in 5 specimens prepared by the agitation method.) Such animals, as a rule, are only carriers. Not so harmless are the worm nodules containing larvæ (Fig. 68) when present in large numbers. They may then produce, according to the number present, a severe or even fatal catarrh of the

small and large intestines by interfering with the activity of the same and by favoring secondary bacterial infection. According to Theiler, a peritonitis frequently develops from the rupture of a purulent worm nodule into the abdominal cavity, but particularly through the multiplication of the microorganisms localized in the worm nodules and their final penetration of the wall of the intestines to the surface of the serosa.

Anatomical Changes. In the small intestine and cecum of cattle, and in the large intestine of other animals, tubercular nodules (roundworm nodules) are found often isolated: frequently also in great numbers, in hundreds and thousands. (Drechsler once counted 430; Marotel saw in one case 4,000 in the small intestine and 1,000 in the large intestine.) The nodules vary from the size of a pinhead to that of a pea, and

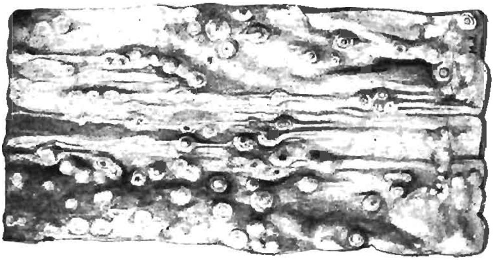


Fig. 68. Worm nodules containing Œsophagostomum larvæ in the small intestine of a cow.

they may occasionally be as large as a hazelnut; the smallest ones have a blackish color, the medium-sized nodules are blackish to grayish-white, the larger ones are always grayish-white. The otherwise unchanged intestinal mucosa shows half-spherical projections: it appears somewhat thinner at this place and in hogs it is also ulcerated.

The thick connective-tissue capsule of the nodules sometimes surrounds a very small cheesy plug, or in large nodules a puslike, sometimes calcified, material. In the more recent nodules there can also be found Œsophagostomum larvæ 3 or 4 to 7 mm. long. Now and then mesenteric lymph glands will be found enlarged and to contain larvæ (Grimaldi found them involved in 15 to 70 per cent of the cases). The mature worms are found in the posterior part of the small intestines and in the large intestines.

Symptoms. Morbid symptoms are only observed in intense invasion and they consist in untractable profuse diarrhea,

progressive emaciation, anemia and cachexia. The disease then usually terminates fatally in coma which comes on after two to three months. When the disease appears as an enzootic one-half of affected animals may die. In South Africa many cases of invagination of the intestine in sheep were observed to be due to the presence of large numbers of worm nodules. According to Theiler, there was also a general peritonitis which rapidly terminated fatally.

Diagnosis. The occurrence of the above mentioned symptoms following sojourn on wet, marshy pastures may suggest œsophagostomiasis nodularis, but a reliable diagnosis can only be made on the basis of some postmortem examinations, or in case worm nodules and mature worms are both present by the microscopic detection of large numbers of eggs in the feces, while on the other hand the detection of only a few eggs in the feces has no diagnostic significance in this respect. In case the microscopic examination is positive, it becomes necessary first to eliminate dochmiasis, in view of the fact that the eggs of Œsophagostomum very closely resemble those of Dochmius (see page 384).

Treatment. Favorable results have not as yet been obtained in the nodular form with the customary vermifuges. According to the assertions of Brumpt, enemas of a watery solution of thymol promise to be very effective against mature worms. Up to the present this has been tried only in sheep (Railliet).

On the previous night the sheep are dosed with 25 to 30 gm. of Glauber salt dissolved in 1 liter of water. On the next day finely powdered thymol is sifted through a cloth bag and made into a paste with a little slightly mucilaginous water. The required amount for each sheep (1 gm. thymol per 2 to 5 kg. body weight) is emulsified with 1 to 1½ liters lukewarm water (35-38° C.). This emulsion is now introduced into the rectum of the animal by means of a funnel or irrigator attached to a rubber tube, during which process the posterior end of the animal is held up and the expulsion of the liquid prevented by pressure upon the anus. After the injection, and while the anus is still being compressed, the abdomen of the animal is thoroughly massaged in order that the emulsion may be carried as far as possible toward the stomach and may thoroughly flush the large intestine. The animal, when released, will stagger about and sometimes lies resting upon the abdomen for an hour. No further disturbances occur, however, and the animal soon expels the killed worms.

Besides the medical treatment, the animals should be well fed. The administration of antidiarrheic drugs (see page 182) may be indicated. The same prophylactic measures are indicated as in strongylosis of the stomach (see page 329). Theiler

recommends rotation of pasture for the lambs or placing them on a pasture that has not been used for at least one year.

Linguatulosis nodularis. Velu observed an enzootic occurrence of this pathologic condition in cattle in Morocco. It is caused by an exceptionally massive invasion, by larvæ of Linguatula, of the intestinal wall, particularly of the posterior portion of the small intestine and of the cecum, with a similar invasion of the mesenteric lymph glands, the liver and the lungs. Flat, hemorrhagic nodules from the size of a pinhead to that of a lentil or a pea, each containing a larva from 4 to 5 mm. long, are formed under the intestinal mucosa and in the muscularis. The nodules are soon changed to small abscesses and thereby become more whitish in color and finally resemble tubercles. Similar cavities communicate with the lumen of the intestine after the larvæ have migrated into the intestinal tract.

The symptoms are not very definite. Diarrhea, emaciation, hydremia with edema of the pendent parts (dewlap, throat) and cachexia are observed. A definite clinical diagnosis is therefore impossible. Upon postmortem, Œsophagostomum must be eliminated.

Treatment is not successful, hence only prophylaxis is indicated. The intestines of herbivora suffering from the disease should be destroyed and dogs harboring Pentastomum should not be allowed to come in contact with cattle.

(h) Strongyloids in the Intestine.

Strongyloidosis intestinalis.

Occurrence. This disease, first described by Reisinger, occurs enzootically in hogs in Austria and leads to severe enzootics in barns. It has also been reported from Germany, England and France. According to Wester, it occurs in colts and hogs. Disturbances of health are observed exclusively in quite young animals (pigs, colts). Strongyloids have also been found in cattle, sheep, dogs, rabbits, the beech marten, the weasel and the polecat.

Etiology. In animals disturbances of health are produced by the parasitic generation of strongyloides, a threadworm belonging to the Angiostomata.

Strongyloides longus. The parasitic form is hermaphroditic, of feminine appearance, with long esophagus, threadlike, 4 to 6 mm. long, the posterior end curved and pointed, the anus in front of the posterior end, the vulva near the last quarter of the body and surrounded by papillæ. According to Reisinger, the eggs (Fig. 69) are 60 microns long; according to Neveu-Lemaire, Fiebiger and others, only 40 microns long, elliptical, and usually contain a coiled, lively moving embryo. Only seldom are they found still in the morula stage. The emerging

rhabditiform larva (Fig. 70) is pointed at the posterior end, only slightly tapering at the anterior end, and has a short esophagus. It is found in the hog, cattle, sheep, rabbit, beech marten, weasel and polecat (Reisinger).

Strongyloides westeri is threadlike, 8 to 9 mm. long. The ova are 45 to 54 microns long, 30 to 45 microns wide, egg-shaped to round, sometimes even polyhedral, and often already contain an embryo or are still in the cell stage (Wester, Ihle). What relationship this worm bears to Strongyloides viviparus (in the large intestine of horses and asses) has not yet been determined.

Fig. 69. Ovum of Strongyloides longus. (After Reisinger.)

Fig. 70. Larva of Strongyloides longus. (After Reisinger.)

Closely related to these worms is Strongyloides intestinalis (Strongyloides stercoralis, Anguillula hominis s. intestinalis, Rhabdonema strongyloides, R. intestinale, Rhabditis stercoralis), parasitic in man and, according to Fuelleborn, identical with the strongyloides of dogs.

Anguillula, which is closely related to Strongyloides, is found widely distributed in nature (water, soil, decaying material, plants), but is only exceptionally found in the intestinal tract of man and now and then upon the skin (see threadworms in the skin).

Evolution of the Strongyloides.

At the time of passage with the feces of the host the eggs of Strongyloides intestinalis have already hatched; the eggs of S. westeri contain embryos, while some eggs of S. longus contain embryos and others have already hatched. Two forms of development take place, according to the outside conditions or possibly the origin of the rhabditiform embryos. Larvæ that were passed with the feces of the host develop in the presence of sufficient moisture and warmth (25 to 28° C.) into a rhabditiform free-living intermediate generation in the outside world; they have a short esophagus with two enlargements (Anguil-

lula, Rhabditis stercoralis); the sexes are separate. Later these develop into a second generation of rhabditiform larvæ which become filariform after molting; or again, the embryos pass through the feces and develop directly into filariform larvæ. After ingestion of filariform larvæ, which alone are capable of infesting a new host, they develop into the sexual parasitic form in 8 days (according to results with feeding experiments by Reisinger); Strongyloides westeri in 15 to 21 days (according to feeding experiments by de Blieck and Baudet).

Resistance. In decaying fecal matter that has later dried out the eggs retain their capacity of further development for several months, while the young forms (embryos, larvæ) of the parasitic generation disappear within a few days. The rhabditic form shows very great resistance and will retain its vitality for more than 6 months in spite of drying out of the feces (Reisinger).

Infestation takes place in pigs as well as in colts in the barn during the first few days of life by their taking up filariform larvæ, which are abundantly present in the soil or in the fecal matter that has collected in the cracks of the floor, with the feed or litter; in pigs also while rooting. The immigration of the larvæ occurs similarly to that of Dochmius larvæ (see page 376), particularly through the skin (filariform larvæ placed upon the well-moistened skin covered with hair are capable of penetrating into the skin), through the walls of the mouth, pharynx, esophagus, and the stomach, in case the larvæ reach the stomach soon enough, while otherwise they are destroyed in the stomach (Fuelleborn, de Blieck and Baudet). They penetrate the skin mostly through the hair follicles, partly also through the microscopic cracks in the epidermis. After the larvæ have penetrated into living tissue they are, in the course of 10 days, carried along by the venous blood by way of the lungs, bronchi, trachea, pharynx and esophagus into the stomach, having in the meantime become almost sexually mature and now being able to withstand the destructive action of the gastric juice. Some of them pass through the capillaries of the lungs and are carried back with arterial blood to the intestinal tract, where they also become sexually mature.

Pathogenesis. Strongyloides have no harmful effect whatever upon grown animals, in spite of the fact that, particularly in grown hogs, they are often present in large numbers (worm carriers). According to the observations of Wester, they do not seem to be able to live in older colts (de Blieck and Baudet could infect even grown horses). In young colts and particularly in young pigs, when taken up in large numbers soon after birth they produce digestive and nutritional disturbances by injuring the mucous membrane of the intestinal tract. By the penetration of the larvæ, microorganisms may be carried along

which may produce a bacterial infection. Upon the passage of the larvæ through the lungs and the bronchi, bronchopneumonia may be produced.

Anatomical Changes. In pigs that have died from the effects of the worms there is found a general anemia, emaciation, eczema of the skin, catarrh of the conjunctiva with opacity of the cornea, often bronchial catarrh with atelectasis, bronchopneumonia, sometimes with a simultaneous necrosis of the lung tissue, and pleurisy. In young pigs sometimes there is also punctiform or blotchy reddening of the intestinal mucosa, which in older pigs may here and there assume a slate-gray color. At the same time a light serous infiltration of the intestinal wall is present. The intestinal contents have a foul odor. The worms in them can be seen only when viewed by transmitted light, in a thin layer between two glass slides, or by microscopic examination, when the eggs and embryos also become visible. In cultures made from feces (see page 341) the larvæ behave similarly to those of the strongyles of the stomach (see page 327).

Symptoms. In colts only a heavy infestation will now and then produce mild colic, emaciation, and sometimes diarrhea. Spontaneous recovery sometimes occurs.

In young pigs, on the other hand, disturbances of health are always produced. In light cases they show a dirty, ungroomed appearance, insufficient development, emaciation, and sometimes eczema, while in severe cases eczema, conjunctival and intestinal catarrh, emaciation leading to cachexia and anemia, and in some of the animals pneumonia, are observed. The disease lasts from 2 to 4 weeks and has a mortality of 0 to 50%.

Diagnosis. This depends upon the detection in the feces of the mature worms and their eggs and larvæ, which may be found within 10 days after infestation or birth. In somewhat older pigs the exclusion of worms in the stomach may become difficult, because the eggs of Spiroptera strongylina and Strongylus rubidus are also passed in the embryonic stage (see pages 332 and 333).

Treatment and Prophylaxis. According to Wester, colts are easily cured with Fowler's solution (5 to 8 gm. daily for a few days). In weaned pigs Reisinger obtained decided improvement with flores cinæ (15 to 20 gm. three times on consecutive days with the feed), while when a severe cough and fever had developed even repeated treatment was fruitless. Marotel recommends extract of male fern (3 to 5 gm. once a week for 3 weeks), thymol (2 to 3 gm. for 3 days), and areca nut (3 to 10 gm. daily for a week).

As prophylactic measures the following are recommended: Cleaning of the stalls, daily removal of the manure, and

)rinkling of the floor of the stalls with milk of lime; also, according to Reisinger, the expulsion of the worms from sows just before or immediately after farrowing.

Other Filiform Worms in the Intestines. Strongylus (Trichostrongylus) tenuis, which lives in the cecum of geese and, occasionally, of ducks and hens, causes no disturbances in health.

Sclerostomum hypostomum (Chabertia ovina) is often found in the colon of sheep, goats, roes, and of deer; it may give rise to intestinal hemorrhage and to diarrhea followed by anemia and emaciation which may cause numerous cases of death among the yearlings. Its eggs, 90-110 μ long, have a similar appearance to those of the other sclerostoma (see Fig. 71) and the evolution of the brood in the outer world takes a similar course. For treatment, Brumpt recommends rectal injections with thymol.

Filaria equina (F. papillosa) is frequent in the abdominal cavity and beneath the peritoneum in horses, asses and mules, and sometimes it is found also in the intestines of horses, without giving rise to any disturbances in health. The filaria embryos, which are often found in the blood in horses are said, by Railliet, to belong to this worm and to be transmitted by insects (Simulia).

(i) Echinorhynchus Gigas of Swine.

Etiology. The **Echinorhynchus gigas** (Gigantorhynchus hirudinaceus) is a thick, cylindrical, white worm, thicker at the anterior end; its external surface appears annulated; an intestinal canal is not present. The protrusible, rounded cephalic end is armed with five or seven rows of strong hoofs (Fig. 72); male 6-9 cm., female 20-35 cm. long; ova (Fig. 73) oval; the embryo is visible within a triple envelope; the head of the embryo is provided with hooklets.

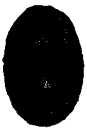


Fig. 71. Egg of Sclerostomum hypostomum.

The following may be said about the development of Echinorhynchus gigas: According to Schneider, the ova are taken up with the larvæ of the cockchafer (May-bug, Melontha vulgaris). The free embryos perforate the intestines of these Arthropodes, become encapsulated in their abdominal cavity and remain alive even in the adult May-bugs. If hogs feed on May-bugs or their larvæ, the capsule of the parasitic larvæ becomes dissolved in the intestinal canal, the larvæ then bore into the intestinal wall of the host. Echinorhynchus larvæ can probably go through their development also in other animals, aside from the May-bug. This seems to be proven by the fact that the worm also occurs in America where May-bugs do not exist. According to Lespes, ech. larvæ occur in certain snails; according to Kaiser and Blanchard, in Cetonia aurata; according to Stiles, in Lachnosterna arcuata. The invasion takes place in such a manner that the larvæ of the Echinorhynchus are taken up by the larvæ of the May-bug or by other intermediate hosts. Young hogs are infected with particular ease and frequently become very ill.

Echinorhynchus is found with marked frequency in some years and then causes great loss among young pigs. It appears as if this periodic frequency stands in relation to the three to four years period of development of May-bugs.

Anatomical Changes. Grayish nodules or yellowish purulent points surrounded by a red halo are seen shining through the serosa of the intestines (Koconrek). The worms bore into the mucosa with their anterior ends; the neighborhood of the mucosa where the parasites are located is intensely reddened and also shows catarrhal swelling. Sometimes the parasites have perforated the intestinal wall and have produced a purulent peritonitis. Sometimes the mucosa shows cicatricial formations.

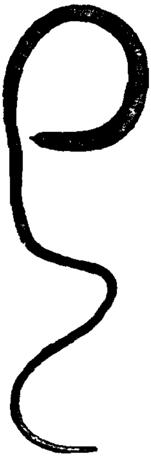


Fig. 72. Echinorhynchus gigas, one-half natural size.

Fig. 73. Ovum of Echinorhynchus gigas.

Symptoms. Digestive disturbances occur proportionately to the number and to the stage of development of the worms; the animals become emaciated and enfeebled. Since the parasites cause pain, the animals grunt continually; they are restless, bury into the ground or straw, snap towards the abdomen or towards other animals. Occasionally, particularly in young pigs, one sees more or less intense twitching of the muscles or epileptiform convulsions, generally followed by a fatal termination.

The cause of these symptoms is revealed, by a microscopic examination of the feces, by the discovery of the characteristic ova; frequently, however, such an examination is neglected and the true cause of the disease is only found on postmortem examination.

Treatment and Prophylaxis. These worms can be expelled only with difficulty. One may try drugs that are effective against ascaris, especially santonin (2-5 gm.), rhizoma filicis maris (20-50.0 gm., or of the extract 2-5.0 gm.), kamala (2-4.0 gm.); also, according to the recommendation of Kocourek, oil of turpentine (in tablespoonful doses, followed by laxatives, see page 516).

The destruction of insects and other animals, which may contain the larval stage of the worm, is indicated as a prophylactic measure, also the avoidance of probably infected parts of land.

Echinorhynchus in Other Animals. Parker found about 300 specimens of the **Echinorhynchus canis** in the intestine of a dog which had died with rabid and paralytic symptoms in the course of 24 hours.

Echinorhynchus in Fowls. According to Railliet, there are probably two species: Polymorphus boschadis (Echinorhynchus minutus coccineus, E. boschadis, E. anatis, E. polymorphus) and Filicollis anatis (Echinorhynchus anatis, E. polymorphus). Up to the present time these have been combined under the name E. polymorphus. They are found in the intestinal tract of ducks, seldom in geese and swans, and sometimes produce enzootics with diarrhea, emaciation and anemia. The eggs are spindle-shaped, 130 microns long and 19 microns wide, and contain an embryo armed with hooks, which develops to the larval stage in beach fleas (Gammarus pulex) and in the river crawfish (Astacus fluviatilis).

30. Protozoa in the Intestines.

(a) Red Dysentery of Cattle. Dysenteria coccidiosa bovum.

(*Kokzidienruhr* [German]; *Entérite hémorrhagique, Flux de sang* [French].)

Red dysentery of cattle is a disease preferably of young animals, generally occurring as an enzootic affection, and due to Coccidium Zürni; these are generally found in the large

intestines, preferably in the rectum. The disease manifests itself particularly by hemorrhagic defecation.

Historical. Proeger (1877) described a disease in calves due to psorospermium, and Zuern (1878) found coccidia in the inflamed intestine of a calf. It was, however, only in 1892, that Zschokke pointed out the etiologic relation between coccidia in the intestines and red dysentery. Hess (1892) and Guillebeau (1893) confirmed the findings of Zschokke, and Guillebeau made culture and inoculation experiments. Others who have studied this disease were Degoix (1904), Züblin (1908), also Bugge, Warringsholz and Sieg (1909).

Occurrence. The disease occurs almost exclusively during the warm season between the months of June and September and especially in wet years; it may, however, be observed here and there later in the fall and even in winter. Commonly (95 to 100 per cent) animals become infected on the pasture and preferably in mountainous parts, more rarely in lowlands.

The disease has been observed particularly in the mountainous cantons of Switzerland, in certain parts of France, but it has also occurred in Denmark (Poulsen), in North Germany, and has been observed in Hungary by Makoldy, also in England, the United States, South Africa, Abyssinia, Tunis, Hungary, Italy, and recently in Holland and British Columbia.

Etiology. Coccidium Zurni (Eimeria) are found in the feces of sick animals as round or oval formations (oocysts) with a long diameter of 10-20 μ according to Zschokke, 18-25 μ according to Degoix, and 15-25 μ according to Züblin; the width

Fig. 74. Intestinal coccidia. (According to Degoix.)

varies between 10-20 μ. De Blieck and Douwes found a small spherical form, 12 to 18 microns long, and a large, more egg-shaped form, 12 to 28 microns long. The feces of a calf infected under natural conditions contained both forms, while in a yearling not manifestly sick only the large form could be found. The highly refractive protoplasm is finely or coarsely granular or globularly contracted (Fig. 74); it shows a light spot in the center (nucleus). There are also seen hyaline folded membranes; these are probably dead coccidia. The membrane of unstained cells appears shining and peculiarly greenish violet in color (small form) or again sometimes more brownish (large form).

The development of coccidia is as follows (Fig. 75): In the oocysts voided with the feces there appear four globular sporoblasts, if the conditions of moisture, heat and access to free oxygen are favorable, within 2 to 6 days; these assume a spindle shape and become sporocysts; in these two crescents or sickle-shaped sporozoites are developed. These changes may occur in 4 or 5, or only in 14 or 15 days, according to environment. Embedded in thick layers of dung or in the deep layer of water, the oocysts remain unchanged without sporulating for from 2 to 2½ months, but retain their capacity to undergo further development. The oocysts are very resistant and can remain alive after putrefactive processes lasting for three months (Degoix). After oocysts have been ingested their mem-

brane is dissolved by the digestive juices, particularly by the pancreatic juice (according to Züblin in 6 to 8 hours), they are set free and then form the lively motile sporozoites which penetrate into the intestinal epithelia and assume a roundish form, being then found between the cell margin and the nucleus. They enlarge gradually and become schizontes, which multiply asexually (schizogony) by forming numerous spindle-shaped merozoites. After the dissolution of the infected epithelial cells these get into the intestinal lumen and invade new epithelial cells; the process of schizogony is repeated several times; some merozoites situated in epithelial cells then change into macrogametes and microgametes. After the latter have become free they penetrate into macrogametes with active motility, fertilize these and form again an oocyst.

The **infection** is spread by the water of pools and marshes since the oocysts, which after ripening are very resistant, are easily carried into these by drainage; or by vegetation growing in the proximity of such places, or by dry feed and drinking water when these have come in contact with the oocysts contained in the feces of sick animals or carriers. The possibility

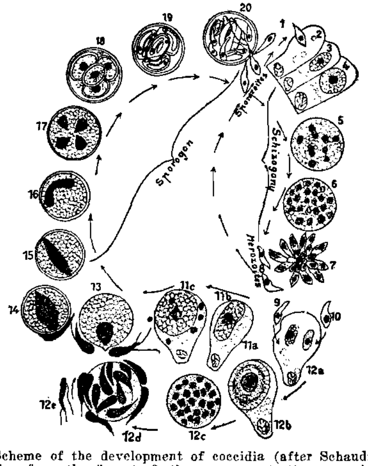


Fig. 75.* Scheme of the development of coccidia (after Schaudinn). 1, Sporozoites passing from the öocyst; 2, the same penetrating an epithelial cell; 3-8, schizogony or asexual multiplication; 8-10, merozoites which penetrate into new epithelial cells; 11a, 11b, 11c, development of the macrogametes; 12a, 12b, 12c, development of microgametes; 12e, free moving macrogamete; 13, impregnation; 14, impregnated macrogamete; 13-20, sporogony or sexual development.

of infection in the barn is suggested by the fact that isolated cases also occur in winter in infected regions, also by the occurrence of coccidiosis among other species of animals which are not pastured, and by an observation of Zürn that calves became infected which had been confined permanently to the barn. Damp and dirty barns undoubtedly are particularly dangerous.

Three young cattle, experimentally fed on material containing sporozoites fell sick after three weeks with the symptoms of acute intestinal catarrh, with diarrhea and a few coccidia in the feces, but without any blood. These symptoms lasted three days (Guillebeau). In the experiment of de Blieck and Douwes a calf infected a few days after birth became sick on the nineteenth day, showing a febrile, long-continued, severe intestinal catarrh and finally a watery, foul-smelling diarrhea without intestinal hemorrhage. Numerous coccidia of the small form were found in the stools. A 1½-year-old cow did not become manifestly sick, although two months later a postmortem examination revealed the presence of patchy thickening of the colon with oocysts of the large form. The disease could not be transmitted by oocysts that did not contain sporozoites. The disease cannot be transferred by non-sporozoitic oocysts. Züblin did not succeed in infecting rabbits with coccidia from cattle that were suffering from red dysentery.

The susceptibility is greatest in youth, up to the second year of life, but older cattle likewise contract the disease, although more rarely. The great rarity of the disease among calves less than six months old may be explained by the fact that these animals are usually not pastured. Isolated cases have, however, been observed among calves three to six weeks old (Zürn, Bugge, Waringsholz & Sieg).

Pathogenesis. The sporozoites escaped from the ingested oozytes, wander with the intestinal contents toward the anus and penetrate into the epithelial cells of the crypts of Lieberkühn of the large intestine, particularly into those of the rectum. They multiply rapidly by schizogony and the epithelial cells of the surrounding glands and those of the mucosa in general are invaded by autoinfection; in this manner an increasing area of the mucosa becomes affected; the usual localization of the changes in the large intestine is attributed by Züblin to a comparatively slow solution of the oocyte membrane and to a rapid passage of the contents of the small intestine into the large intestine. It is, however, remarkable that the small intestine is also profoundedly affected in young calves and that the small intestine of other species of animals is most profoundly affected.

After the epithelial cells have become invaded they are shed and this produces a denudation of the glands and of the mucosa, and the congested capillaries protrude free towards the intestinal lumen. This explains the occurrence of intestinal hemorrhages. At the same time irritation by coccidia produces a small round cell infiltration of the mucosa and of the layers below it. Since the mucosa becomes denuded in spots and deprived of its protecting covering, an intense invasion of debilitated animals may lead to secondary infection by intestinal bacteria and this may produce severe enteritis and general infection.

Anatomical Changes. One usually finds the intestines contracted; in the large intestine and particularly in the rectum one finds thin fluid greenish or reddish-gray, to reddish-brown contents, sometimes containing distinct blood coagula or even ichorous masses. The mucosa of the rectum is usually affected most profoundly, the mucosa of the colon or cecum much less

or not at all, although the latter sometimes shows profound changes. The mucosa shows strong corrugation, reddening, swelling and a mucoid, fibrinoid, yellowish or grayish deposit sometimes mixed with blood. In consequence of the loss of epithelial cells the surface of the mucosa appears rough in places with hemorrhagic spots or streaks. Epithelial losses and floating shreds of mucus or detached epithelial cells are seen in advanced cases, alternating with slate gray spots of the mucosa. Necrotic processes in the large intestine with catarrhal or hemorrhagic inflammation in the small intestine are

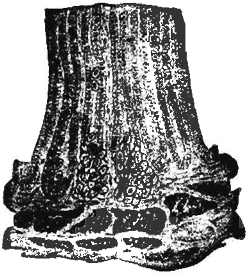


Fig. 76. Position of intestinal coccidia in the glands of Lieberkühn. Low magnification. (According to Degoix.)

frequently seen in consequence of a secondary infection. Numerous coccidia (Fig. 76) are seen in the intestinal contents and in detached shreds of the mucosa. The cadavers also exhibit the signs of profound anemia and cachexia.

Symptoms. The peculiarities of the development of coccidia bring it about that the period of incubation of the disease is comparatively long, about three weeks; occasionally, however, symptoms may come on after six or eight days (Bugge, Waringsholz & Sieg). On the basis of his observations Degoix claims that the period of incubation is one or two months; he does so because he saw the disease come on in cattle after they had been in the barn for one or two months.

The disease begins immediately with diarrhea and after one or two days the feces show an admixture with blood and mucus; distinct blood coagula from the size of a walnut to that of a child's fist are also seen; the feces are fetid and sometimes have a cadaverous smell. As soon as hemorrhagic feces appear, there is at first slight, later on intense tenesmus, which may lead to prolapse of the rectum. In other cases normal feces are

voided at first, followed by a small amount of coagulated blood, later on feces and blood are mixed and diarrhea supervenes.

In this stage mild cases soon pass into recovery, particularly in adult cattle; the blood disappears after two or three days, and after a few more days the diarrhea stops. The appetite, however, remains capricious for some time.

In grave cases, particularly in young animals, there is a rapid deterioration of the condition. Tenesmus becomes more severe, the feces become quite fluid, very fetid; they contain larger coagula and shreds of mucus; appetite and rumination are suppressed entirely. At the same time the emaciation progresses rapidly and the gait of the animals becomes staggering. The pulse rises gradually to 70-80 and later on even higher; the temperature rises to 40° C. and above. Later on the blood disappears from the feces, but they often contain lumps of mucus, like croupous membranes, and an ichorous thin fluid. The emaciation has now reached a high degree, the eyes are deeply sunken, the animals can no longer stand up and they die from complete prostration.

Course and Prognosis. The disease runs an acute course of five to ten days, but there are also hyperacute cases in which the animals die within twenty-four hours in convulsions. The appetite sometimes remains poor for a while after disappearance of the grave symptoms, so that the animals become much emaciated; anemia may then become so intense that the patients may die even after five months. In the mild, favorable cases, recurrent attacks are quite frequent, but these usually take a benign course. Very mild cases lead to the symptoms of simple acute intestinal catarrh.

As complications of the disease one can sometimes observe blackleg, convulsions and coma in pregnant animals, bronchitis and pneumonia.

The prognosis is the more unfavorable, the younger and the weaker the sick animals; the more rapidly the appetite decreases, the earlier rumination ceases. Two to four per cent of the sick animals usually succumb to the affection.

Diagnosis. The occurrence of the disease during the time of pasturing, the almost exclusive affection of young animals, hemorrhagic diarrhea and the rapid emaciation suggest the diagnosis. The detection of the coccidia by microscopic examination of the feces makes the diagnosis absolute and excludes rectal hemorrhage of cattle (see page 170). The diagnostic significance of the detection of coccidia in the feces is greatly reduced by the fact that H. Mueller found in the intestinal tract of 42 cows oocysts or the young stage of coccidia in 88% without finding anatomical changes at the same time (carriers). Also Velu found coccidia without manifest symptoms in mature cattle in Morocco, the animals merely acting as carriers, as was

found by Noeller and Frenz at the Berlin abattoir, where 7 out of 12 calves and 3 out of 16 cows harbored coccidia.

Coccidia are at first found in the diarrheic stools only sparingly; they appear, however, in large numbers in the shreds of mucus and they can, according to Züblin, be seen in the voided coagula. In the terminal stages of the disease the number of coccidia again decreases. In order to find the coccidia more easily Züblin recommends staining microscopic cover-glass preparations with Lugol's solution; Bruce, the following enrichment method: The feces are passed through a wire sieve and then pressed through several pieces of linen of varying density; the fluid last obtained is centrifuged for several minutes, the upper column of fluid decanted, and the sediment examined for oocysts. The sodium chloride method is the best.

Treatment and Prophylaxis. Pasturing should be interrupted and dry feeding instituted, if the disease appears in a herd. Disinfectants (creolin, lysol, resorcin, sodium thio-sulphuricum) have been used as long as the disease has been known; also astringents (tannic acid, silver nitrate, iron sulphate) in the form of rectal injections in weak solutions, or they may be given as a mucilage or with milk by the mouth. Züblin saw good results from rectal injections with 1 per cent tannic acid or 1 per cent alum.

Sieg also obtained splendid results from the administration of tannin. Ott saw good curative results following the internal administration of the following mixture: Creolin Pearson 20, tincture rhei aquæ 40, tincture calami 40, once a day. In three cases reported by Saulorezo a complete cure was rapidly effected by the administration of thymol (15 gm. daily). Italian veterinarians report good results with thymol. Salvisberg observed a cure even in hopeless cases after the administration of the following mixture: Tannoform, bismuth subnitrate āā 50, animal charcoal, powdered cocoa āā 250 (3 times daily 6 gm. of this mixture on the tongue of the animal; in larger animals the amount of the animal charcoal should be increased 4 to 5 times). Galli-Valerio suggested chemotherapy with atoxyl and sodium antimonyl tartrate (sodium emeticum), which could also be used in the form of injections in which atoxyl and tartar emetic are combined. This treatment was successfully used against trypanosomes in mules by Ruppert (every tenth to eleventh day subcutaneous injection of 20 cc. of a 10% solution of atoxyl, then on two other consecutive days an intravenous injection of 2 gm. tartar emetic at blood heat after it has been dissolved in 25 cc. hot water). If the animals suffer from lack of appetite, they must be fed with milk and eggs (Salvisberg saw an increase in the digestive disturbances following the administration of mucilaginous drugs or milk). Severe intestinal bleeding should be arrested.

As a prophylactic measure the ingestion of dirty water from pools and marshes must be prevented; if the disease makes its appearance, the healthy animals should receive dry feeding, or they should at least be brought to dry pastures where they have access to pure water.

By soaking the feces in a 5% solution of cresol compounds (acids in 3 to 5% solutions are ineffective, according to the experiments of Grosse) such as creolin, lysol, betalysol, cresotin-cresol, grotan and desintol, the coccidia are said to be killed. In case of an enzootic in a barn the stalls should be kept dry and clean, and in case new breeding animals are purchased care should be taken to avoid carriers of the infection. (See also prophylaxis in coccidia of sheep and goats.)

(b) Intestinal Coccidiosis of Sheep and Goats. Coccidiosis ovium et caprarum.

Occurrence. Intestinal coccidiosis frequently occurs in sheep and goats, either sporadically or enzootically, particularly in the early summer and in young animals, and not infrequently causes heavy losses.

According to Moussu, the disease appears in France every year in flocks of sheep of different origin, as in Germany, where it occurs enzootically in goats and sheep, particularly in several breeding flocks as well as elsewhere (Karsten, Honeker, Spiegl, Lerche, Grosse). It was furthermore observed by Wester and Beyers in native sheep and hogs. It also occurs in Hungary in goats and in Morocco enzootically in sheep.

Etiology. In sheep Coccidium Faurei (Eimeria Faurei) is found, also, according to Lerche, Coccidium Arloingi. The oocysts of Coccidium Arloingi are round or elliptical, in the latter case 21-36 microns long and 16-25 microns wide, with sickle-shaped, helmet-like, or button-shaped cap at the pole at which the microphyle is located. The oocysts of Coccidium Faurei occur in a larger egg-shaped form with a maximum length of 42 microns and width of 30 microns, and in a smaller roundish form with a diameter as small as 17 microns.

Fig. 77. Coccidium Arloingi. Oocyst from a goat.

Infection takes place on low, wet pastures, through the vegetation, and probably also through drinking water. It also frequently occurs in the barn, under influences that decrease the resistance of the animals and favor infection with a rapid multiplication of the coccidia, such as faulty weaning, cold (after shearing), complete changes of living conditions (as in change of place, placing the animals in another flock, sudden changes of feed, feed of a poor quality, especially in time of war), faulty feeding, unfavorable weather conditions, inbreeding, and the youth of the animal. Under such conditions a

latent coccidiosis may suddenly become acute. This is indicated by the observations that in Hungary a few coccidia are frequently found in kids and adult goats without manifest symptoms (Marek), and that Grosse found 70 kids from different regions of the province of Saxony and Anhalt infected with coccidia, and in 40 flocks of sheep a varying number were found to harbor coccidia without showing manifest symptoms. Coccidiosis is mostly introduced into a flock by seemingly healthy carriers, seldom by manifestly sick animals. It seems that sheep may also become infected from goats (Ifland reports that coccidiosis was introduced into a flock of goats by an emaciated sheep suffering with diarrhea).

Anatomical Changes. These are found especially in the small intestines (Wester and Beyers saw changes throughout the entire intestinal tract with numerous coccidia in a lamb), the mucous membrane of which is more or less closely studded with gray to yellowish-white finely granulated nodules from the size of a pin point to that of a pin head (corresponding to the places where the coccidia have penetrated the epithelium). A light reddening is also often observed; more frequently, however, only in mucous catarrh with a slimy discharge. Oocysts or young stages of the coccidia are found in the intestinal contents or in scrapings of the mucous membrane. Honecker found in a kid numerous coccidia in the grayish-white gall ducts, together with yellowish, cheesy, smeary masses of mucus. The anatomical picture is supplemented by the phenomena of severe anemia, emaciation and ascites, and by streaky hemorrhages in the swollen nasal mucosa.

Symptoms. After a period of incubation of 2 to 4 weeks, in spite of a fairly good but rather mostly depressed appetite there will be observed anemia, emaciation, foul-smelling diarrhea, usually without bloody discharges, and extreme weakness. Convulsions may later also appear. After from a few to several weeks the animals die from exhaustion. Many kids and lambs, however, die in a few hours following the sudden development of bloating and great weakness. Such animals utter peculiar cries and bleat. In protracted cases nasal and conjunctival catarrh also occur. The mortality may reach 90%. The animals which recover remain carriers for months.

Diagnosis. This depends upon the anamnesis, the presence of stubborn intestinal disturbances, and especially upon the microscopic detection of numerous coccidia in the feces, while only a few oocysts are of little significance in this respect. A positive diagnosis on the basis of clinical symptoms is impossible, especially the exclusion of dysentery of lambs, hemorrhagic septicemia of sheep, worm disease and a primary anemia.

Treatment and Prophylaxis. No curative drug for coccidiosis has yet been found, although Wester and Beyers saw

recovery in several lambs after they were placed on higher ground and were given 4 gm. salol and 8 gm. tannoform daily. Other drugs such as are used in coccidiosis of cattle (see page 409) may also be tried. In some of the author's observations an improvement took place after the administration of 0.5 to 1 gm. atoxyl and 0.25 to 0.5 gm. tartarus stibiatus alternated daily.

The main efforts must be directed to a proper prophylaxis. Particularly should dry feed be given in order to prevent further infection with oocysts. For this reason, the animals at pasture should be immediately stabled and the pastures closed to other susceptible animals for 6 months. If this is impracticable the pastures should at least be changed and the animals placed on higher and drier ground. The animals remaining healthy should be separated from the infected ones (under favorable conditions they should be stabled separately). The repeated examination of the feces for coccidia will insure the timely recognition of coccidiosis in infected and in threatened flocks. The intestinal discharges and the bedding should be removed daily from the stalls and covered with straw and a layer of soil (the oocysts will be destroyed by the spontaneous heating), or be burned or placed on fields containing much lime, but in no case should they be placed on fields from which green forage is to be fed. The animals may also be changed daily from one stall to another. The vacated stall should at once be thoroughly cleaned of all dung, swept with a stiff broom, and disinfected. According to Oppermann, it will suffice to remove the top layer of dung to a depth of 10-20 cm. from sheep stalls in which mattress bedding is used, and to cover the lower layers with a 5 cm. layer of sand or peat which has been saturated with a suitable disinfectant. A kind of permanent disinfection by using chlorinated peat as bedding is also recommended. For the daily cleaning and disinfection of the stalls at short intervals—a procedure that is especially recommended—cresol compounds have been found most reliable (see page 410). All parts of the stall that have come in contact with the excretions of the animals, including the mangers, feed buckets and boxes, should first be cleaned with a hot soda solution. In goat stalls the animals should be prevented from lying down in the feed troughs by the proper placing or nailing of strips across the top. The ground of exercise lots or barnyards should be covered with a thick layer of milk of lime and then spaded up. A further prophylactic measure consists in burning or burying deeply all cadavers and organs of sick animals. As measures to increase the resistance of the animals, the sucklings should be gradually and properly weaned and properly fed and cared for, and colds and inbreeding should be prevented.

(c) Intestinal Coccidiosis in Pigs.

Neveu-Lemaire was the first to report the occurrence of

Eimeria Jalina in the cecum and the large intestine of pigs. The investigations of Douwes, and especially those of Noeller and Frenz, indicate that coccidia are quite frequently found in the intestines of pigs. (In Holland they are quite prevalent in young pigs. At the Berlin abattoir they were found in 75% of 20 fecal specimens examined by the sodium chloride method.) Even grown hogs harbor them, and although normally they do not show any symptoms, yet the sows act as carriers and spread the disease to the young pigs, which die of coccidiosis in large numbers (Noeller and Frenz observed it in 10-day old pigs). Intestinal coccidiosis, therefore, seems of some significance as a disease of young pigs.

The symptoms of the manifestly sick animals consist of a severe, acute intestinal catarrh. On postmortem there is found injection of the blood vessels and a catarrhal or even hemorrhagic inflammation of the intestine, especially the jejunum.

While Douwes distinguished two forms of coccidia of different size (50x33 microns and 24x18 microns) and attributes a pathogenic effect only to the larger, Noeller and Frenz found all the intermediate forms between the larger (36x26 microns) and the smaller, and believe it not impossible that all coccidia in pigs belong to Eimeria suis, as described by Noeller, which forms spores in 10 to 14 days (at room temperature).

(d) Intestinal Coccidiosis in Carnivora.

The coccidia found in the intestinal villi of dogs and cats belong to Coccidium bigeminum (Isospora s. Diplospora bigemina). As a rule, they do not produce any morbid symptoms. Dages found in the epithelium of the intestinal mucosa coccidia which, according to Martin, appear to be identical with Coccidium cuniculi. Jakob observed a more or less severe intestinal catarrh and loss of strength in several dogs that passed numerous coccidia in the feces. In the Institute for Tropical Diseases at Hamburg, Noeller found all young dogs and cats, in particular dogs 4 weeks old, infected with Isospora. Deaths occurred especially in the cats.

(e) Intestinal Coccidiosis in Rabbits. Coccidiosis intestinalis cuniculi.

Occurrence. Coccidiosis is widespread in rabbits and usually occurs as hepatic or intestinal coccidiosis. Either form occurs separately or both occur together; seldom are they found together with nasal coccidiosis. Sustmann frequently found all the stock, or all young animals between 6 and 8 weeks old, infected.

Etiology. All types of coccidiosis in rabbits are caused by Coccidium oviforme s. cuniculi (Eimeria Stiedæ). The egg-shaped oocysts have a relatively thick shell and occur in two sizes. The smaller form, which is 26-35 microns long and 14-20

microns wide, is found especially in the intestinal tract (also called Coccidium perforans), and the larger form, which is 40-49 microns long and 22-28 microns wide, is found particularly in the gall ducts.

Infection is spread by feed or drinking water which has been contaminated with oocysts from the feces of sick or apparently healthy animals, and is favored by the ingestion of green feed, particularly when too much is fed, and by weakening influences (sudden change of temperature or feed, long railroad transportation, poor condition of the animals). Young animals are especially susceptible, and it is these for the most part which

Fig. 78. Oocyst from Coccidium cuniculi.

become manifestly sick, while older animals kept under the same conditions are less easily infected and then as a rule do not show any morbid symptoms.

Anatomical Changes. Anatomical changes are usually found in the small intestines and consist of an intense intestinal catarrh with the accumulation of tenacious, slightly reddened mucus upon the swollen and moderately reddened mucous membrane, which latter is studded with whitish nodules not larger than a lentil. Sometimes the changes are very slight.

Symptoms. These are either identical with those of hepatic coccidiosis (which see) or the infection runs the course of a violent enteritis with exhausting diarrhea, especially in young animals, and is followed by death in a few days. In cases of manifest intestinal symptoms the mortality reaches 90 to 100%.

Diagnosis. Diagnosis is made by the microscopic detection of very numerous oocysts in the feces or by postmortem examination. Hepatic coccidiosis differs clinically only in the presence of relatively few oocysts of the large type.

Treatment and Prophylaxis. As in the case of other animals, no curative drug is known for coccidiosis in rabbits. As in intestinal coccidiosis in general, a treatment similar to that for acute intestinal catarrh (see page 182) is indicated, but favorable results are usually not obtained. Ottolenghi and Pabis have nearly always achieved a cure with atoxyl and sodium emeticum (the administration of 0.04 to 0.08 gm. atoxyl and 0.006 gm. sodium emeticum alternately every 2 days for 2 weeks), but in practice this treatment does not seem to have given satisfactory results. Neither have satisfactory results followed the administration of medicinal methylene blue (1 gm. in 4 liters of boiled water as drinking water, or twice daily a tablespoonful of a 0.4% solution). According to Sustmann,

fairly satisfactory results can be obtained with glycerin or with a powder made up by him (a mixture of disinfecting, enveloping and astringent chemical and vegetable products). In cases of a simultaneous distention of the intestines with gases, Seebrandt obtained good results with barium chloride (0.2 to 0.4 gm. in 100 cc. distilled water, a tablespoonful twice daily).

In the face of these facts, prophylactic measures seem to be imperative. For this purpose measures similar to those recommended in coccidiosis in sheep and goats (see page 414) may be used, with necessary changes. Racbiger and Spiegl recommend that the floor of the hutches be constructed of tightly stretched wire netting with meshes large enough for the feces to drop through, with a box half filled with pulverized peat under the same; also that the young animals be separated from the old as soon as possible. Sustmann recommends that the young be left with their mothers only long enough to nurse, and that they be kept in a separate cage during the rest of the time. Greeu feed should not be allowed (especially for young animals). Other necessary measures consist in the removal or destruction of all manifestly sick or runty animals; the separation of the healthy animals from those that have been found to be carriers by the examiuation of the feces (if possible there should be daily examination of the feces of infected stock); exclusion of carriers for breeding purposes, and keeping and feeding them separately till ready for slaughter; increasing the resistance of the animal by allowing predominantly dry feed with the addition of a limited amount of green feed or turnips; protection from cold; protection from troublesome bucks and from external parasites; construction of proper runways; addition of calcium salts (precipitated chalk, calcium chloride, cracked bone meal) to the feed; avoiding undernutrition in the sucklings by removing the supernumerary young, and placing a resting board for the doe half way up in the cage so that the young can not reach her. Dead animals, infected entrails, and the feces should be destroyed either by burying them or by burning them after they have been soaked with kerosene. The cages should be cleaned daily and kept dry (by using straw, hay or peat as bedding), and all utensils should also be cleaned daily. The latter should be disinfected (see page 412) at least every 2 weeks. The use of feed racks constructed of wire netting and of feed troughs with a strip or wire netting nailed over it lengthwise (to prevent the animals from crawling into them) should also be considered. The greatest care should be exercised in buying new breeding stock. The feces should be examined on the first day and, if necessary, also on the fifth to sixth day following, and all animals found infected should either be refused or kept and fed separately and later slaughtered. All shipping boxes and other utensils that can not be easily disinfected should be burned.

Coccidiosis in Guinea Pigs. According to Bugge and Heinke, coccidiosis in guinea pigs, which formerly occurred but seldom and only in the light form, has increased very much under the influence of the bad feed conditions during and after the World War, so that many caviaries are now probably infected. (In 73% of the guinea pigs from different places, oocysts of coccidia 16-25 microns long and 12-17 microns wide were found in the feces.) Clinical significance and treatment are similar to coccidiosis in rabbits.

(f) Intestinal Coccidiosis of Fowl. Coccidiosis intestinalis avium.

(*Weisse Ruhr des Geflügels* [German].)

Occurrence. Intestinal coccidiosis is observed among all species of domestic fowl, also in pheasants and grouse, particularly among young animals, which die in large numbers at the age of 4 to 12 days. The birds sicken after they have reached the age of at least 1 month.

Etiology. Coccidium tenellum (Eimeria avium) invades the intestines of fowl. Different species of coccidium described as Coccidium Pfeifferi (pigeons), C. avium (chicken and ducks), C. truncatum (geese and ducks), C. oviforme (pheasants) are probably all only varieties of Coccidium tenellum.

Natural infection occurs with food or with other objects taken up by fowl (sand, mud, etc.) and with the drinking water.

(Hébrant and Antoine found numerous oocysts of coccidia in the grain feed and drinking water of infected flocks.) The infection is spread by carriers, particularly by infected fowls, but also by those that show no symptoms, or those that have recovered from coccidiosis (especially grown birds).

Eckardt looks upon coccidia as facultative parasites, which during summer multiply rapidly in remnants of food or other organic material that are taken up accidentally by birds, and which then further develop in their intestines. This view is supported by an observation of Kleinpaul, who saw an affection of ducks which had been kept in a dried marshy swamp.

The spreading of fowl coccidiosis might occur by the aid of eggs laid by sick birds. Eckardt found coccidia on the shells of eggs and in their albumen. The same observations had been made previously by Podvissotzky. Sjöbring doubts that the formations found in the eggs were coccidia, while C. Curtice denies the possibility of the transmission of coccidiosis through the eggs.

The infection experiments of Railliet & Lucet and Eckardt were positive in 2 or 3 weeks. In Eckardt's experiments symptoms of disease came on within 3 to 6 days and the disease took either an acute or a chronic course. Fautham infected chicks and young pigeons with oocytes from grouse; Jowett infected chicks with Coccidium cuniculi.

Anatomical Changes. These are found in a portion of the cases in the cecum, and in another in the small intestines (in the woodhen, especially in the duodenum), while in other cases the greater portion of the whole intestinal tract is affected. In the acute form of the disease are found changes of an intense

acute intestinal catarrh or of enteritis, with patchy desquamation of epithelium, the intestinal contents are dirty grayish-white, possibly also bloody; the mucosa presents grayish-white nodules or spots up to the size of a millet seed.

Sometimes, particularly in geese, the intestines contain masses of fibrin. In cases of very short duration, the inflammatory changes may be entirely absent, so that the cause of death is only demonstrated by finding under the microscope great numbers of coccidia in the intestinal contents (Kleinpaul), and in which cases the real cause of death may be a septicemia due to bipolar or other bacteria penetrating at the point where the epithelium of the mucous membrane has become desquamated.

In subacute or chronic cases the intestinal mucosa of the emaciated animals appears reddened in spots, coated with tenacious mucus and as though dusted with flour. Such dustlike points occasionally occur also in the liver; they are then also due to coccidia (Eckardt).

Symptoms. Violent uncontrollable diarrhea, listlessness and increased thirst exist in the **acute form**, while the appetite at first remains normal. Then there occurs profnse salivation, drowsiness, lack of appetite and bluish discoloration of the comb. The droppings become slimy, possibly hemorrhagic.

Meyer and Crocker sometimes also observed discharges from the eyes and nose, also a special conjunctival form of the disease with fibrin deposits upon the conjunctiva and cornea.

Death occurs after two or four days, but sometimes, particularly in chicks and ducks, suddenly without any premonitory symptoms. In Eber's case the chicks had bled to death into the ceca.

The **subacute form** manifests itself in similar symptoms as the preceding type; its course is, however, slower; the animals remain alive two to four weeks and become emaciated so that they are finally reduced to mere skeletons.

The **chronic form** has been seen in experimental animals and in natural infection in pheasants; it leads to the development of a marantic condition and diarrhea may alternate with constipation.

According to Eckardt the mortality is 60 to 70 per cent; sometimes all young animals affected die.

Diagnosis. The disease can be diagnosticated only by the microscopic discovery of the coccidia in the droppings (Fig. 79) or in the intestinal contents of dead animals; the described clinical symptoms in connection with the history may excite a proper suspicion. The diagnostic value of the microscopical demonstration of coccidia in the droppings is somewhat diminished by the fact that, according to Cole and Hadley, as many as

90% of chickens, and many turkeys in some regions, discharge coccidia with their droppings, without manifesting any symptoms of disease (carriers). For this reason the proposal of Hébrant and Antoine to undertake a microscopic examination of scrapings from the intestinal mucosa in doubtful cases seems justified, since in the presence of pathogenic coccidia, the young stages (schizonts, merozoites) can be found within the intestinal epithelium. The differential diagnosis has to consider fowl cholera, chicken plague and the various septicemic diseases of birds, and in turkeys infectious enterohepatitis.

Treatment. Treatment promises little success. Morse recommends the administration of 0.006-0.01 gm. of calomel or a few drops of castor oil, with two or three drops of oil of turpentine. Railliet and Lucet prescribe sodium subsulfurosum (0.3-2 gm.) for chickens; Klee for pigeons 5 gm. sulphate of iron and 15 gm. pure glycerine in one quart of boiled water.

Meyer and Crocker found these and other drugs of no value, and with medicinal methylene blue (see page 414) they could only delay death from 5 to 6 weeks. In the woodhen Fantham saw good results from catechu (0.2 gm. to 1 liter of drinking water for at least 10 days). The chemotherapy of Ottolenghi and Pabis (see page 414) could also be tried.

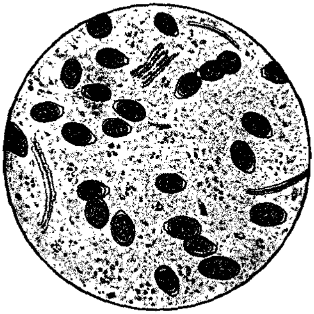


Fig. 79. Coccidium avium in the intestinal tract of a young goose.

Prophylaxis. The separation of the sick from the healthy animals must at once be provided for, then thorough disinfection of the coops and daily thorough cleaning. The barnyard where fowls are kept should be kept dry, the droppings should be removed, the incubators should be disinfected and dried out, the floors calcimined and these spaded up to a depth of 45 cm. The ground of the runways should also be treated with finely powdered iron sulphate (100 to 150 gm. per square meter), the grain feed thoroughly washed, wet mashes prepared with only

boiled water, the drinking water boiled, and if possible the young chicks reared on new dry ground.

Morse recommends purgation of the hens one or two weeks before they begin laying, and cleaning of the eggs that are to be hatched, with 95% alcohol.

Water fowl should be kept away from half-dried out pools or swamps. Eckhardt succeeded in stopping an enzootic of coccidiosis among chicks by giving them sterile water and sand sterilized by heat.

Extensive experiments on the value of milk feeding in changing the hydrogen ion concentration of the contents of the ceca of chickens and its bearing on the effect of milk feeding on artificial infection with Coccidium tenellum (Eimeria avium) have been conducted by Beach and Davis at the California Agr. Exp. Sta.

The range of hydrogen ion concentration of the cecal contents of fowls fed the usual grain ration was found to be from 6.0 to 7.4. The hydrogen ion concentration was increased to a range of from 4.4 to 5.6 by feeding sufficient amounts of whole sweet milk, milk cultures of B. acidophilus, milk cultures of B. acidophilus plus lactose, lactose alone, or dry skim-milk. The change in the hydrogen ion concentration appears to be due entirely to the lactose in the milk products.

The increase in the hydrogen ion concentration from a single feeding of a milk product occurred within two to two and one-half hours after the feeding and returned to normal within eight to twenty-four hours after the feeding. The increase in the hydrogen ion concentration was constantly maintained by the individual administration of one or two grams of lactose twice each day or by the continuous feeding of mash mixtures containing 20% lactose or 40% dry skim-milk. Feeding chickens mash mixtures containing sufficient lactose or dry skim-milk to cause a constant increase in the hydrogen ion concentration of the cecal contents gave them a considerable degree of protection against artificial infection with sporulated oocysts of **Eimeria avium.** A mash mixture containing dry skim-milk was more effective in this respect than one containing lactose. This difference appears to be due in part to the greater consumption of mash containing dry skim-milk by the chicks and in part to the superior food value of this milk product.

(g) Trichomonas in the Intestine of Fowls. Trichomonasis intestinalis avium.

(Ansteckende Blinddarm-Leberentzuendung der Truthuehner, Schwarzkoepfigkeit, Flagellose [German]; Enterohepatitis infectiosa, Amibiasis meleagridum; tête noire, crise rouge [French]; blackhead [English].)

Infectious Inflammation of Cecum and Liver of Turkeys. Entero-hepatitis infectiosa meleagridum.

Occurrence. The affection occurs particularly in North

America and is known as "Black-head"; it has been observed also in Europe, Africa and Australia. This disease attacks young turkeys, by preference, especially those from three weeks to two or three months old, particularly at the time of the development of the comb and wattles and during the molting season, since the birds are more susceptible at this time. It causes heavy losses in flocks of turkeys but also occurs in other species of fowls.

Several diseases have been designated as "Black-head," more particularly entero-hepatitis, intestinal coccidiosis, helminthiasis, cholera of fowls, and in general diseases with dark discoloration of the comb and wattles of the affected animals.

Etiology. According to the results of recent investigations by Hadley, the disease is caused by Trichomonas, belonging to the flagellates, which is found in a nonparasitic form in all species of fowls and under special conditions has a morbid action.

Infection occurs by taking up the droppings of sick or apparently recovered birds (carriers), soiled feed, and contaminated water. V. A. Moore succeeded in reproducing the disease in healthy turkeys by feeding the diseased organs, and Tizze and Faby by injecting emulsified liver foci subcutaneously, whereupon the lungs, liver, kidneys and intestines became diseased. It has not been proved that the disease can be transmitted by the eggs. An infection is not absolutely necessary, since the saprophytic flagellates originally in the intestinal tract may, in case of a weakening of the body (as by diarrhea, heterakiasis and other forms of helminthiasis, molt, etc.), penetrate into the intestinal tissue and there multiply.

Pathogenesis. In healthy birds the Trichomonas organisms live in the contents of the ceca, particularly in the layer of mucus next to the epithelium, and frequently also at the base of the glands of Lieberkuehn. They multiply in the intestinal contents by division; however, the usual mode of propagation appears to be by encystation and spore formation within the tissue, where, to be sure, ameboid forms are also met with which coincide with the form designated by Smith as Amœba meleagridis and with certain bodies at one time believed by Hadley to be stages of evolution of coccidia.

The motile flagellates multiply in the contents of the ceca, invade the goblet cells at the base of the glands of Lieberkuehn, and finally reach the submucous connective tissue. The invaded epithelial cells degenerate, thus opening a way for the further penetration of the flagellates. Those forms which have penetrated into the tissue soon develop into roundish or ovoid bodies which stain with eosin and which rapidly multiply by encystation and spore formation, thus producing a thickening of the intestinal wall. After the destruction of the intestinal epithelium their escape into the lumen of the intestine is made possible.

Some of the motile flagellates, migrating through the epithelium, are probably carried away by the blood stream of the portal vein into the liver and localize in the cells of this organ. Here they may be found together with Amœba intestinalis, which has later penetrated into the liver. In the ceca they may also be found with coccidia.

Anatomical Changes. The cecum is enlarged uni- or bilaterally, its walls are thickened and its lumen is obstructed, in part, by reddish intestinal contents and by exfoliated necrotic tissue. The mucous membrane is covered with several layers of fibrinous membranes and with necrotic tissue, in some portions it is destroyed by ulceration. The liver is enlarged to 2-3 times its normal size; it contains numerous yellowish, firm foci with softened centers, of which the superficial ones appear to be slightly depressed, at times.

Similar foci are sometimes found in the kidneys (Kaupp, Charmoy). Sometimes adhesions are found between the ceca and other intestinal loops, between the liver and the muscular portion of the stomach, and also between the liver and the glandular portion of the stomach. Numerous flagellates are found under the microscope (especially in a hanging drop) in the tissue of the mucous membrane, in the glands of the ceca, and in the necrotic foci of the liver.

Symptoms. After an incubation period lasting at most one month, the disease commences with loss of appetite, listlessness and weakness, soon followed by diarrhea with ill-smelling, yellowish, thin-fluid evacuations. At the same time the animals rapidly become emaciated; in most cases the comb and wattles and the non-feathered portions of the skin show a dark-gray or blackish discoloration (hence the English name "Blackhead").

The animals die toward the end of the first, or at the beginning of the second week; occasionally the disease assumes a chronic course in older animals and leads to a surprising degree of emaciation. The mortality is as high as 80-90% in young animals, and about 20% in adult animals. Convalescence usually requires a long time and may be interrupted by a relapse. The apparently recovered animal continues to evacuate Trichomonas.

Treatment and prophylaxis are similar to those in intestinal coccidiosis of birds (see page 418), but here, too, treatment usually remains unsuccessful. According to M. and P. Wegeforth, 29 out of 32 sick birds recovered after the administration of fluid extract of ipecacuanha (10 drops 3 times daily for 3 days, then twice daily for 3 days, and finally once daily for 3 days). The disease is said to be suppressed also by feeding powdered ipecacuanha in a wet mash (1 teaspoonful twice a week for 10 birds). Emetin should also be tried (0.02 to 0.03

gm.). Smith recommends the following prophylactic measures: Artificial incubation, separation of the young poults from older birds, periodical change of the location of the houses, frequent inspection of the flock, and isolation of the weak and listless birds and their destruction in case improvement does not take place in a few days, otherwise they are afterwards to be placed with the older birds.

Amebic Dysentery in Animals. Amebic dysentery has been observed in a badger (Melos taxus) in the zoological garden of Cairo, in an orang-outang in Manila (Kartulis), and in other monkeys in America (Eichhorn and Gallagher). Recently true amebic dysentery has also been described in dogs, particularly 1 case by Kartulis, 8 cases by Ware in India, and 1 case in Madras by Bauche, Motais and Gauducheau. This is supported by the observation of Gauducheau, according to which a dog contracted severe amebic dysentery after taking up intestinal discharges from a man suffering with dysentery, as did another dog after the introduction of intestinal mucous from a sick man. Weakening influences seem to be of some significance, for, according to the observation of Bauche, Motais and Gauducheau, a 1½-year-old nursing bitch contracted amebic dysentery.

Besides more or less pronounced debility in some cases, the symptoms consist in bloody mucous diarrhea containing red and white corpuscles, numerous trichomonas-like flagellates and numerous ameba in the intestinal discharges. On postmortem, ulcers are found in the large intestines.

In the cases of Ware a dietetic treatment combined with the administration of emetin (0.03 to 0.06 gm.) effected a cure.

Amebae in the Intestines of Other Animals. Lehmann described a case of disease in horses, under the name of ameboid dysentery, in which a horse had succumbed after four weeks of illness attended by slight symptoms of depraved appetite. On autopsy the mucous membrane of the small intestine was found exfoliated, of honeycomb appearance, with many ulcers, 5-6 cm. in diameter with thick borders and smooth bases; also thickening of the intestinal walls, and in places necrosis of the mucous membrane and round-cell infiltration of the intestinal wall. Beneath the surface of the mucous membrane the tissues contained numerous formations looking in form and size like an ameba. Lehmann is inclined to attribute the necrotic crusts which are found occasionally in the fore-stomachs of cattle likewise to the action of ameba-like formations which are found underneath the crusts in the tissues, and also an intestinal disease in lambs associated with the formation of papillomas on the intestinal mucosa, in which buttons develop, up to 3 mm. high and 3-5 mm. wide, with narrow base and having the consistency of the intestinal mucosa. The results of Lehmann, which were obtained only on material that had been preserved, require confirmation by the examination of fresh material.

Ameboid Dysentery in Man. This disease, which is known also by the name of Tropical Dysentery, occurs endemically, occasionally

epidemically in tropical and subtropical regions, and at times in the temperate zones; it is caused by the amebae of dysentery (Entamoeba histolytica and E. tetragena), in contrast to the bacillary dysentery **which** is due **to the** action of bacteria. Ameboid dysentery can be produced artificially in dogs and particularly in young **cats, by** introducing the feces containing amebae of affected humans deep into the rectum. In this manner it can be shown that the amebae invade the glandular ducts of the mucous membrane, and further penetrate the submucosa, particularly in the colon, where they give rise at first to a hemorrhagic catarrhal inflammation of the mucous membrane and swelling of the solitary follicles. In the further course of the affection, a gradual thickening of the mucous membrane, destruction by ulceration, and the formation of extensive tortuous and sometimes deep losses of substance with undermined borders take place, also the formation of fibrinous exudate and single or multiple abscesses in the liver.

At first the symptoms consist in colicky pains, tenesmus, diarrhea with raspberry-jelly-like evacuations with contain much blood and mucus; at first the general condition is affected but little and there is no fever. With the development of ulcers, bloody stools appear which have a cadaver-like odor and are mixed with shreds of mucous membrane; there is frequently vomiting and moderate fever. A suitable mode of treatment often leads to recovery in 1 to 2 weeks, but in many cases a chronic invalidism persists.

The diagnosis is established by the demonstration of the amebae in the mucus or in the pus-masses of the stools, in fresh cover-glass specimens or particularly in the hanging drop. In contrast to the harmless entameba coli, the amebae of dysentery show no nuclear membrane and present a separation of the ento- and ektoplasma.

The treatment consists in rest in bed, suitable diet, and the administration of calomel and astringents (Koske & Hetsch, Die Exp. Bakteriol. und die Infektionskrankh., Bd. II, Wien, 1911).

(h) Flagellates and Infusoria in the Intestine.

Of the **Flagellates,** *Lamblia intestinalis* (*Megastoma entericum,* Cercomonas intestinalis), which is supplied with a suction-cup, usually lives in the small bowel of mice and rats, also in rabbits, more rarely in dogs, sheep and cats. In dogs and cats it may cause a fatal gastroenteritis (Sirtirana, Perroncito) and seems to multiply also very much in inflammatory intestinal diseases of other origin. Trichomonas suis occurs in the intestines of hogs, Trichmonas columbae in the intestines of pigeons, and Monas anatis in the large bowel of ducks and of chickens. Spirocheta canis seems to be a common but harmless inhabitant of the stomach in dogs, but appears to multiply greatly in inflammatory diseases of the mucous membranes.

Of the **Infusoria,** above all the Balantidium coli is to be mentioned, a very frequent inhabitant of the colon in hogs, which does not cause any disturbance in health, but is merely an intestinal saprophyte similar to the numerous varieties of infusoria in the colon of the equidae, also in the rumen of ruminants.

SECTION VI.

DISEASES OF THE LIVER.

1. Jaundice. Icterus.

Under the term jaundice is understood the passing of bile substances into the lymphatic system, into the blood, and thence into the tissues. As the result of a granular deposition of bile pigment, the body tissues and excretions are stained yellow.

Etiology. Especially in dogs, jaundice is caused most frequently by catarrh of the duodenum (Icterus catarrhalis) the outflow of the bile being hindered through the swelling of the mucous membrane at the outlets of the liver duct or the bile duct; at times the mucosa of the larger bilary canals may be involved. Accordingly, jaundice is frequently observed in the course of acute gastroenteric catarrh. According to Chierici, colon bacteria, which reach the biliary ducts from the uninjured intestine, may produce jaundice by causing a primary catarrh of the bile passages (infectious jaundice). Of rare causes are to be mentioned: invagination of the duodenum, tumors of the pyloric region (Eberlein), obstruction of the biliary passages by foreign bodies, parasites (clumps of liver-flukes, roundworms), gallstones, tumors, cicatricial adhesions. In a similar manner the compression of the biliary passages by enlarged periportal lymph nodes may act, also echinococci, new-formations near the porta hepatis, enlargement of adjoining organs, and also chronic pyelophlebitis. Jaundice which makes its appearance in the course of acute or chronic hepatitis and of passive hyperemia of the liver is due to a compression of the smallest biliary passages. According to Chierici superficial losses of substance in the intestinal mucosa may afford an opportunity for colon bacteria to get into the blood of the portal vein and, with it, into the liver; they will then cause immediate injury to the liver cells and thereby give rise to infectious jaundice. Schmidt observed severe jaundice in a horse affected with torsion of the left lobe of the liver.

Thrombosis, compression of the portal vein, owing either to enlargement of the pancreas or of the portal lymphatic glands, and chronic inflammation of the portal vein are very exceptional causes of jaundice.

The immediate cause of the jaundice observed in cases of poisoning by phosphorus, arsenic, salt, lupines, etc., is to be found in the plugging of the minute primary bile ducts with débris of liver cells.

Not rarely hemoglobinemia (due to piroplasmosia, extensive burning of the skin, and certain infectious diseases, etc.) is associated with jaundice. In such cases excessive quantities of bile pigment are manufactured by the liver cells from the abnormally large amount of blood pigment that is present in solution in the blood plasma (pleiochromia), while there is not a proportional production of the fluid constituents of the bile. The bile produced under these circumstances is viscid and is with difficulty passed out of the liver, consequently a portion is reabsorbed.

The form of jaundice that sometimes occurs during attacks of acute infectious diseases, severe inflammation of the lungs, and hemoglobinemia arising from unknown causes, was at one time termed hematogenous jaundice. Recent investigations have shown that the bile pigments in these cases also are produced by the liver.

It is not definitely settled whether in individual cases of hepatitis accompanied by jaundice the diseased liver cells do not pass a proportion of the bile direct into the blood capillaries (paracholia, parapedesis).

Icterus neonatorum makes its appearance especially in newborn calves and foals shortly after birth or, exceptionally as a congenital condition; sometimes it passes off in a few days, but in the majority of cases it causes death. The cause for this affection was assumed, by Hartmann, to be an inflammation of the umbilical vein, but in most cases the jaundice has another origin, and arises probably more particularly through pleochromia induced by a free disintegration of red blood cells, by the absorption of non-broken down bile-coloring matter into the blood of the portal vein, which is then transported into the general circulation through the Ductus Arantii which is open for the time being, also possibly because of a general debility and insufficient respiration. It is not certain whether an injury to the liver cells may stand in relation, induced by the sudden increase of their activity at the beginning of taking food.

According to Ylppo, it is merely a special form of parapedesis, and the partial passage of bile into the blood represents merely a fetal activity of the liver of short duration following birth, which, during fetal life, continually sends part of the bile pigments into the blood.

Pathogenesis. Aside from icterus neonatorum and icterus hemolyticus, jaundice can always be traced to a stasis of the bile which is produced by mechanical obstruction in diseases of the bile duct, the liver tissue and the duodenum, or to a thickening of the bile in case of superabundant production of bile pigments in the course of hemoglobinemia. In case of thickening of the bile this causes the formation of plugs (bile thrombi of Eppinger, liver cylinders of Affanasiew) which obstruct the fine biliary capillaries and consist of deposits of bile pigment or coagulable protein material. In all such cases the bile is dammed back toward the liver and distends the biliary passages back to the intercellular biliary capillaries, the terminals of which are distended and allow the escape of bile, which is under increased pressure. The bile is now permitted to escape into the neighboring lymph capillaries and probably also into the blood capillaries which are separated from the lymph vessels only by a thin membrane consisting of Kupffer's cells. Sometimes the walls of the greatly distended biliary capillaries burst, whereupon the bile can pass directly into the lymph and blood capillaries (Eppinger, Umber).

Anatomical Changes. With the exception of the nerve tissue all the organs are stained yellow as a result of the absorption of

bile pigments. This is most marked in tissues that contain fat, such as the mesentery, the fat capsule of the kidneys, subepicardial fat and subcutaneous connective tissue. It may, however, also be observed in blood clots, in any fluid exudates that may be present, and in the muscular tissue. (Rüther states that in birds affected with jaundice there may be a greenish pigmentation of the peritoneum.)

In the horse the fat is always yellow. In other animals it is also more or less yellow under certain conditions of diet and even when the animals are fed on certain pastures. In emaciated animals the subcutaneous connective tissue is sometimes saturated at places with yellow serum. The simultaneous yellow discoloration of all the tissues renders the diagnosis of jaundice easy.

Symptoms. A characteristic feature of jaundice is the yellow coloration of the mucous membranes and of the unpigmented parts of the skin. This is recognizable in the earliest stages in the scleral and palpebral portions of the conjunctivæ. It is less distinct in the mucous membranes of the mouth and nose, unless one causes a temporary anemia of the membrane by exerting pressure upon it. The color varies with the severity and duration of the disease from a lemon- to an orange-, or even a greenish yellow tint. Unpigmented parts of the skin appear, as a rule, rather more intensely stained.

The condition may be recognized still earlier by an examination of the urine which is either orange-, brownish-, or greenish-yellow in color, and if agitated gives a yellow froth. White filter paper, linen or silk are stained by the urine. Chemical tests show the presence of bile pigments and often also of bile acids, while microscopic examination of the sediment reveals the presence of masses of pigment which give the bilirubin reaction.

Gmelin's test is one of the best for the chemical demonstration of the presence of bile pigments. In this test the suspected urine is added to some nitric acid containing a few drops of nitrous acid so that it forms a layer above it. A positive result is indicated by a play of colors where the liquids meet, green being the most characteristic. Rosenbach's modification of this test appears to be a practical one. A piece of filter paper moistened with the urine, or better still the paper through which the urine has been filtered, is placed upon a white porcelain slab and is touched with a glass rod that has been dipped in fuming nitric acid. If bile pigments are present a ring of colors appears around the spot where the acid has been placed. These colors from within outwards are yellow, violet, brown, green. In some cases only the green color is obtained.

A very sensitive test is that of Grimbert, in which 10 cc. of urine are mixed with 5 cc. of a 10% solution of barium chloride; shaken vigorously, then centrifuged or filtered. The precipitate is mixed with 4 cc. of 90 % alcohol containing 5 volume-per cent. of hydrochloric acid; heated for one minute after which the liquid assumes a blue- or dark-green color on standing. If a brownish coloration occurs, 2 drops of peroxide of hydrogen are to be added; after heating again, the green color will be clearly manifest.

Bile-pigments tend also to make their appearance in sweat, milk, and exudates, but they are completely absent from the lacrimal secretion and the saliva.

The appetite is suppressed, and emaciation with debility appears if the jaundice persists for some time, probably on

account of the disturbances in the hepatic function. Not infrequently the pulse rate is diminished and at times arrhythmia is noted which may, in part, be produced by the absorption of bile acids. A more or less marked dullness is never absent, associated with fatigue. Albuminuria is frequent, partly due to cholemia, partly to the toxic or infectious substances which produce the jaundice.

As a result of obstruction of the bile-duct the intestinal contents contain little or no bile, and are therefore either pale yellow or gray in color and contain a large quantity of fat. In pleiochromic jaundice, on the other hand, the intestinal contents are stained yellow with bile.

In severe cases of icterus, the symptoms of the so-called Icterus gravis are often observed, viz: hepatic autointoxication, hepatotoxemia; primarily nervous disturbances like listlessness, narcolepsy or loss of consciousness; also local or general muscular convulsions, manifestations of excitement, dyspnea and, at times, Cheyne-Stokes respiration, also symptoms of collapse which may cause death immediately.

The symptoms of icterus gravis are due only in part to the excess of bile substances in the tissue fluids (cholemia), in greater part they are probably brought about through the diminished ability of the liver cells to bind the poisons of protein putrefaction; they may also be attributable to the nitrogen-containing precursors of urea and possibly to the cleavage products of intestinal protein-digestion.

Levrier, Bernadin and others have described a disease as icterus gravis which is observed in newborn mules, and Dieckerhoff saw the same affection in a newborn blooded foal; the course of the affection was that of an infectious disease associated with jaundice and sometimes with bloody micturition and may perhaps be explained as a septicemia due to infection of the umbilicus.

Kohnhäuser has observed five cases of typhoid jaundice in the dog. The symptoms were as follows: dullness, inappetence, vomiting, accelerated pulse, temperature up to 41° C., shallow and painful respiration, yellow discoloration of the mucous membranes, feces small in amount and pale yellow in color, bile pigments in the urine and intense thirst. Later the jaundice became more intense and the animals emitted an unpleasant sweet odor. In two cases there were edematous swellings. The animals died in from 3 to 6 days from complete exhaustion. Chieri has recorded an outbreak of similar cases in which he believed the cause to be a general infection with colon bacilli. It is possible that these were cases of hemoglobinemia due to piroplasma with secondary jaundice.

Course. The prognosis is most favorable when the cause of the jaundice is catarrh of the duodenum or large bile ducts. Occasionally gallstones produce a temporary jaundice. Acute and chronic diseases of the liver parenchyma and chronic inflammatory lesions about the portal fissure cause a more lasting and serious condition, as do also certain forms of poisoning (phosphorus, arsenic, common salt), and independent infections.

Diagnosis. In view of the fact that jaundice is merely a symptom of various organic diseases, the cause of the engorgement with, or absorption of bile must be thoroughly investigated.

Treatment. As a rule good results follow treatment similar to that adopted in gastric catarrh (see page 131).

In addition to careful regulation of the diet, from which all fat is rigorously excluded, the administration of a course of neutral salts is advisable. For dogs a 10 per cent lukewarm solution of Carlsbad salts may be employed. Frequent flushing of the rectum with large quantities of water has a beneficial effect. In order to dilute the bile and to stimulate the contractions of the gall bladder, bile ducts and the intestine, Cozessa advises the subcutaneous injection of pilocarpine in doses (for dogs) of 0.005 to 0.01 gm., followed by two or three doses of 5 gm. of potassium tartrate. Mild purgatives, such as calomel and aloes, are indicated where there is constipation, and in cases in which there is oliguria diuretics such as lemon juice and potassium acetate or nitrate. According to Siedamgrotzky, faradization of the liver is beneficial (?). Horneck has had good results from the intravenous infusion of physiological salt solution.

2. Gallstone Disease. Cholelithiasis.

Occurrence. Apart from precipitations and incrustations in the bile ducts, true gallstones are of rare occurrence. They are, however, found most frequently in the horse and ox, more rarely in the dog, and exceptionally in the cat, pig and sheep. Kitt found a calculus in the gall bladder of a fowl.

Etiology. The basis of gallstone formation is often given by catarrh of the biliary passages, which arises from a primary bacterial infection, most frequently from the intestine through the bile duct, but may also be due to animal parasites (liver-flukes) and to foreign bodies (portions of the intestinal contents) which have entered the biliary passages from the intestine; in this event a subsequent infection is very likely and probably is of importance. General infectious diseases may also give rise to secondary infection of the biliary passages, from the liver, the intestine or by way of the blood stream. Finally, stasis of bile of any origin favors the separation of the solid bile-constituents, as does also the development of an inflammation of the mucosa of the biliary passages because in the case of biliary stasis the microorganisms which accidentally enter the biliary passages can multiply more easily.

Formation of Gallstones. The views concerning the origin of biliary calculi are not yet uniform. According to the explanation of Lichtwitz, gallstone formation begins with the forming of a nucleus consisting predominantly of condensed and coagulated colloid (protein, mucin, nucleoalbumin) and, embedded in this, bilirubin calcium. The precipitation of bilirubin calcium is brought about by the unavoidable disappear-

ance of the colloid protection for the weakly soluble salt compounds of bile and lowering of their solubility, due to the condensation of the colloids, whereupon the weakly soluble salts will precipitate upon the condensed colloid. Such a nucleus continues to act as an active surface foreign to the bile, upon which absorbable colloids will continue to deposit, which, in turn, are again condensed and precipitated, thus causing further deposition of precipitates and an increase in the volume of the concretion, the supporting structure of which is formed by the precipitated colloid. Concrete bodies (cells, eggs of parasites, foreign bodies) may also act as foreign surfaces for the colloids of the bile. Such colloid or condensation concretions (dark brown, white, gelatinous) are sometimes found in the gall bladder of hogs. By encrustation following abundant diffusion into the gall bladder, and deposition of amorphous precipitates, the true, concentrically stratified gallstones are formed. The radially stratified cholesterin gallstones are formed by the deposition of crystals of cholesterin under certain conditions in a radiating direction. The first impulse in animals for the formation of a nucleus is probably a catarrhal condition of bile ducts, induced on the one hand by the occurrence of abundant, easily condensed colloids (protein, mucin) and desquamated epithelia or other concrete bodies which also may furnish the basis for a nucleus, or on the other hand by a change in the solubility of the salts dissolved in the bile as the result of increased formation of cholesterin by the epithelium and increased excretion of calcium, and also as a result of other changes in the chemical composition of the bile. Stasis of the bile, on the other hand, probably plays a primary rôle much less frequently. According to Aschoff, the impulse originates in the liver cells, in which blackish to brownish pigments are formed as a result of stasis of the bile, and after their excretion into the bile ducts coalesce into a clotted mass which forms the nucleus for gallstones in the gall bladder.

Anatomical Changes. The granular precipitates mentioned above are found very often on the mucosa of the bile ducts and in the bile, in the course of liver-fluke disease in ruminants, occasionally upon other fonndations in horses. In old cases these masses form rigid, hollow cylinders attached to the walls of the bile ducts. In horses, cattle and hogs the bile ducts sometimes contain hard cones or cylinders which may be as thick as a finger. In the fresh state they are light or dark brownish-yellow in color and smooth, and when dried, rough and laminated (Kitt). In other cases they are white and composed of chalk-like masses containing minute particles of food. Gurlt found such a stone weighing five pounds.

True gallstones are usually found in larger numbers in the principal bile ducts, or more frequently in the gall bladder.

They are either rounded with mulberry-like rough surfaces or polyhedra with smooth facets, yellowish-brown, greenish, gray or reddish-brown in color. The majority are not very hard and have a specific gravity slightly higher than that of water (1.23). Their cut surfaces shows laminæ of different colors enclosing a more or less soft center.

Small stones varying in size from a linseed to a pea are sometimes found in very large numbers. Birnbaum found 400 in the bile ducts of a horse, Rigot 90, Avérous more than a thousand, Lewin 500 to 600. Single stones, on the other hand, sometimes attain the size of an apple (Verheyen). In Avérous' case there was, in addition to the numerous small stones, a large one measuring 11½ cm. long and weighing 258 gr. in the terminal portion of the principal duct. Messner found a stone weighing 542 gr. in an ox.

The chemical composition of the stones varies from case to case, and especially with regard to the quantity of cholesterin. In gallstones obtained from horses Fürstenberg found lime, mucus, fat and bile pigments. In cattle the stones contain a comparatively large proportion of carbonate and phosphate of lime and magnesium. Bourgoin found a stone weighing 330 gr. in the bile duct of a horse. In this case the stone was composed entirely of cholesterin.

Impaction of gallstones in the common bile duct leads to the production of an inflammatory, hyperplastic cirrhosis of the liver. Occasionally abscess formation results. In other cases there is dilatation of the bile duct associated with atrophy of the surrounding liver tissue. In cattle the common bile duct sometimes attains a diameter equal to that of the large intestine. In such cases peritonitis due to the rupture of the dilated ducts may be the immediate cause of death (Cagny, Chaissaing).

According to Lewin gallstones are sometimes found in the intestine, these having been passed out of the bile ducts.

Symptoms. The gall bladder may contain a large number of gallstones without there being any disturbance of the animal's health. Eberhard found the gall bladder quite full of stones in a pig that was apparently in perfect health. Since gallstones obstruct the outflow of bile the chronic symptoms to which they give rise are not characteristic. The animals usually have a capricious appetite. Offensive diarrhea and constipation alternate. In ruminants there is tympanites, dullness, gradual wasting and anemia, without any symptoms enabling one to determine the cause of the condition.

The sudden impaction of a large gallstone in the common duct or in the neck of the gall bladder produces cramp-like contractions of the wall of the duct. In this way horses and occasionally cattle are attacked with colic-like pains (gallstone colic). These pains may persist with remissions for some days and then disappear. The colic is sometimes associated with loss of consciousness (Birnbaum, Lewin).

On the second or third day of the attack, and in some cases earlier, there is biliary pigmentation of the mucous membranes which appears suddenly with the onset of pain, and disappears as quickly when the pain ceases. In cases in which the neck of

the gall bladder is obstructed there may be no evidence of jaundice. During the attacks of colic there is a rise of temperature. The pulse may be either rapid and small or slow and full, and not rarely irregular. In addition to the constipation and diarrhea vomiting is seen in the dog. Wyssmann observed symptoms of hemorrhagic nephritis in a horse.

In man, attacks of colic are observed occasionally in consequence of an inflammatory irritation of the mucous membrane, or because of the obstruction of the lumen by products of inflammation, and through the swollen mucous membrane. In animals, manifestations of colic may be absent even after the gallstones are wedged in, or there may be only dullness, icteric discoloration of the mucons membranes, disturbances of appetite and of fecal evacuations, also at times sensitiveness to pressure, and enlargement of the liver.

The obstruction of the bile passages not infrequently leads to death through hepatic autointoxication (see page 427) or as rapidly as in one or two days, from perforation-peritonitis after rupture of the gall-bladder or of the bile ducts; in this case a sudden diffuse sensitiveness to pressure becomes apparent, on the part of the abdominal walls, also rapid respiration, a small and hard pulse (Mollereau, Cagny).

Diagnosis. Gallstone disease is recognized only in the presence of gallstone colic together with jaundice, tenderness on pressure, and enlargement of the liver, also manifestations of *Icterus gravis. Frequently recurring symptoms of jaundice* and transitory painful enlargements of the liver permit the supposition that gallstones are present. In some cases the finding of gallstones in the feces make a certain diagnosis possible. It is uncertain at present whether the examination of the fecal evacuations for biliary coloring matters and their products, as suggested by Cozette, can be of deciding value.

Treatment. During the attacks of colic, narcotics, such as morphia, injected subcutaneously, chloral hydrate per os or per anum, and inhalation of chloroform are indicated. Mild purgatives, such as olive oil, are likely to assist in moving the stone by causing an immediate increase in the flow of bile. Parascandolo removed a gallstone from the bile duct of a dog by laparotomy, recovery being established in four weeks.

For the prevention of the formation of fresh stones, moderate diet and plenty of exercise associated with an extended course of neutral salts are indicated.

Foreign Bodies in the Liver and Bile Ducts. Occasionally pointed foreign bodies perforate the reticulum or abomasum and penetrate into the liver. This as a rule leads to the formation of an abscess in the liver and the animal presents the symptoms of traumatic gastritis and purulent hepatitis. Saint-Cyr found the straw of a leguminous

plant in the liver of a horse. Mégnin records a case in which he found some awns of barley which had obviously come from either the stomach or duodenum lying lengthwise in the gastro-hepatic ligament. In the first case, the straw perforated the portal vein and caused thrombosis, and in the second, death was due to hemorrhage. On three occasions Cadéac and Blanc found a needle in the liver of dogs. One of these showed symptoms resembling those of rabies.

In the bile-ducts of oxen, pigs and horses large quantities of sand are sometimes found. This is due to the ingestion of dirty food. In a case recorded by Augenheister the greatly dilated bile-ducts contained 10 lbs. of sand. Kinsley observed no disturbances in health in such hogs, although the bile ducts and gall bladder were not rarely filled with gallstones and dilated after killing, and although jaundice existed. Bits of food are not infrequently forced from the duodenum into the bile ducts and there cause inflammatory changes. Kohlhepp found fecal material in the greatly dilated gall bladder of a cow which had died suddenly.

3. Fatty Liver. Hepar adiposum.

(Fatty Degeneration and Infiltration of the Liver.)

Etiology. The following factors may lead to an increase in the fat content of the liver: Very rich diet in the process of fattening, which is best seen in birds, insufficient exercise, diminished oxidation in the body brought about by anemia. On the other hand, the liver often becomes fatty when there is sudden wasting of an animal as in diabetes mellitus and in certain diseases which run a rapid course. In these cases large quantities of fat are reabsorbed from the fat-containing tissues by the liver, and then gradually consumed.

In many cases fatty liver is caused by noxious chemical materials which are either absorbed from the alimentary canal or are circulating in the blood. The principal of these are the bacterial toxins, fatty liver being a frequent lesion in acute infectious diseases. Acute yellow atrophy of the liver is apparently due to bacterial toxins. Certain poisonous plants produce the same effect. Fatty liver may be associated with gastroenteritis, due to the ingestion of mouldy fodder. The effect of lupines in this connection is remarkable. Of the mineral poisons arsenic, antimony, lead and phosphorus are the principal causes and especially the last. The chief organic compounds that cause fatty liver are carbolic acid and alcohol.

Pathogenesis. According to recent investigations, all forms of fatty degeneration of the liver arise exclusively or mainly through the deposit of fat, or of substances which go to form fat, into the liver cells directly from the food or from the fat-deposits in the body; in cases associated with injury to the liver cells, only a subordinate importance may attach to local fat-formation from the cellular albumin, and to the liberation of the latent fat which is normally present, as well as of the

lecithin. In forced feeding for fattening, in true obesity and in case of rapid emaciation which is not produced by poisonous substances, the fat is deposited in the normal liver cells, which then functionate normally in spite of their fat-content or are disturbed to a certain degree in their nutritional function only in case of excessive deposits of fat. In contrast to this simple fatty infiltration, the degenerative or necrobiotic (Joest) fatty infiltration, which usually is designated as fatty degeneration, origiuates in a primary injury of the liver cells with a simultaneous reduction of their ability to metabolize the fat which is carried to them or is independently synthesized. In part the liberation of the normally latent fat and lecithin may stand in relation. The formation of a portion of the fat, during the disintegration of the cell-protoplasm is denied by F. Mueller and by Pflueger. A necrobiotic fatty infiltration of the liver, with associated functional disturbance of the liver cells, takes place when the oxygen is used up insufficiently in the course of severe anemia with insufficient supply of oxygen, and more frequently under the influence of bacterial or chemical poisons which hinder the protoplasm from making use of the oxygen.

The experimental proof for the deposit of transported fat, in any case of fatty liver, was supplied by Rosenfeld, Lebedeff and Leisering. In the livers of dogs which had been rendered poor in fat, then fed with mutton fat, and finally poisoned with phosphorus, Rosenfeld and Lebedeff found almost exclusively a fat identical with the mutton fat; Leisering failed in his attempts to produce fatty liver in very thin chickens by phosphorus poisoning.

Anatomical Changes. The liver appears pale-yellow and in severe cases may be as yellow as butter or ochre. In the early stages the peripheral parts of the lobes are yellow. In advanced cases the entire liver is intensely yellow in color. The absence of a greenish tint excludes the possibility that the discoloration is due to bile. The liver is enlarged (Neyraud saw a horse's liver that weighed 14 kilos; and Kitt, a pig's liver weighing 14.8 kilos), the edges are rounded, the consistency is decreased, it feels greasy to the touch and pits made by pressure with the finger persist. On cutting into it the blade of the knife is found to be covered with a layer of fat. In cases in which the protoplasm of the liver cells is involved the liver may be actually smaller than normal on account of the absorption of the destroyed liver cells. Evidences of this destruction can be seen under the microscope.

Symptoms. A fatty condition of the liver may be suspected when the predisposing factors are in operation and the liver is enlarged without there being any other symptoms of disease of that organ. When the liver is enlarged there is an increased area of hepatic dullness, and the edges of the organ can be palpated. If the animal be very fat it is generally impossible

to ascertain whether the liver is enlarged but the general condition of the animal makes it extremely likely that the liver is fatty. In the horse, digestive disturbances and particularly constipation are generally observed.

Occasionally fatty livers rupture and the animals die suddenly as a result of internal hemorrhage. There is sometimes slight abdominal pain (Neyraud). Neale records a number of cases in Shropshire sheep which ended fatally after a short illness and in which the only lesion found at the postmortem was extreme fatty infiltration of the liver. Rupture of the liver is especially frequent in well-nourished fowls and geese (Johne).

Treatment. The treatment must be adapted to the nature of the case.

4. Amyloid Disease of the Liver. Degeneratio amyloidea hepatis.

(*Hepar amyloideum, Amyloidosis hepatis.*)

Etiology. Amyloid of liver always develops secondarily in protracted and exhausting diseases, in prolonged cases of suppuration (particularly caries of bone), and in cases of long-continued action of various bacterial toxins; it is most frequent in horses. Bohl found it in 4% of all horses that were autopsied, on the other hand, Joest, Pflug, Foerster, Paulicky, and Hissbach have referred to its very infrequent occurrence in domestic animals. Amyloid of liver has been noted particularly in horses in the course of long drawn out pleurisy and in chronic inflammations of the serous membranes in general in which Rabe has shown the existence of amyloid in almost 50% of the cases. Further primary affections are protracted bronchial catarrh, chronic pneumonia, glanders, in exceptional cases obstruction of the biliary ducts (Bourgoin).

E. Noyer observed amyloid degeneration of the liver followed by rupture of that organ in about 20% of horses used for the production of diphtheria serum. Grüner found amyloid liver in every one of 46 horses that had died from contagious pleuro-pneumonia. On the other hand the very rare occurrence of amyloid liver in the domesticated animals has been recorded by Joest, Pflug, Förster, Paulicky and Hissbach.

Rabe observed the condition in a bitch in connection with carcinoma of the mamma; Bruckmüller, in cattle, as the result of tuberculosis and chronic nephritis, and Ries in lymphangioitis.

Pathogenesis. The nature of amyloid degeneration is not yet entirely elucidated. According to Krawkow's investigations the amyloid may be supposed to form an ester-like compound of albumin with chondroitin-sulphuric acid, which appears to be present in the normal arterial walls according to Minkowski, but cannot be demonstrated directly by microchemical means. According to Ribbert, an unused albuminous substance gets into

the circulation under certain pathological circumstances, it is preferably deposited in the arterial walls and changed into amyloid by uniting with chondroitin-sulphuric acid. On the other hand, it is possibly due to a disturbance in the disintegration of proteins in the tissue cells which are injured through cachectic conditions or through certain poisonous substances; in this case the imperfectly disintegrated proteins would be transformed into amyloid (Wichmann). It is possible that the development of amyloid represents different phases of a protein transformation.

Krawkow succeeded in producing amyloid degeneration experimentally in dogs, rabbits, chickens and pigeons, by repeated subcutaneous injections of cultures of Staphylococcus pyogenes aureus; while culture-extracts of the same bacterium failed to produce the same effect; but systematic injections of toxins of the Bac. pyocyaneus did produce amyloid degeneration. Similar experimental results were obtained by Maximow in rabbits and chickens, by Davidson in rabbits, guinea-pigs, mice and chickens; Ravenna in white rats. Lubarsch was able to produce amyloid degeneration in dogs in 4 weeks, in rabbits in 3 to 4 weeks, by systematic injections of oil of turpentine and by the suppuration produced in this manner; similarly Schepilewsky succeeded more recently with enzymes (pancreatin, papaiotin).

According to Frank, amyloid degeneration is caused by the toxins of an encapsulated bacillus. The bacillus becomes established in necrotic cavities, mostly following a secondary infection, very seldom primarily in bronchopneumonia foci. Extensive amyloid degeneration of the spleen has been produced with this bacillus in white mice in from 8 to 10 days.

Anatomical Changes. The liver is the organ that is most commonly affected. In cases where the degeneration is extensive the liver may be enlarged three to four times. The edges appear thick and rounded, and the capsule is often considerably thickened. The color varies from light brown to yellow or gray. The lobulation is distinct because the peripheral part of each lobule is pale grayish-red and lardaceous, the central portion being pale brownish-red, grayish-brown, or occasionally yellow. The consistency is more or less doughlike, and in the later stages crumbling like half-dried mortar. In birds amyloid liver is granular and brittle. In the early stages no abnormalities save slight enlargement, distinct lobulation and pale color are observed.

On microscopical examination the deposition of amyloid is found around the blood capillaries and in the walls of the blood vessels, but not in the liver cells and connective tissue cells; only Johne observed a simultaneous amyloid degeneration of the liver cells in a horse. In horses the degeneration commences in the periphery of the liver lobules and progresses gradually toward the center; in consequence the union between the individual lobules of the liver is lost early, and in this manner the organ loses its consistency and becomes friable.

Amyloid material may be demonstrated in affected organs by its color reactions. The application of Lugol's solution for a few minutes to the cut surface stains the degenerated parts mahogany-brown. The same test may be applied to microscope sections, the color being changed to a dirty violet or bluish-red by the addition of 2% sulphuric acid. Safranin gives an orange-yellow color.

Symptoms. In cases where there is or has been some primary diseased condition which experience has shown is likely to be followed by amyloid degeneration, a diagnosis of this condition may be based upon the increased area of dulness in the region of the liver and upon palpation of the liver. The symptoms are obscured by those of the primary disease, but as a rule, such symptoms as anorexia, irregular defecation, dulness, wasting and albuminuria, may be due to amyloid disease itself.

The disease is rarely associated with ascites or jaundice. Rupture of the softened liver and resulting hemorrhage are often observed whether there be any traumatic influence in operation or not. In addition to anorexia and dullness, Rexante observed an intermittent bright yellow coloration of the feces which were passed in small quantities only. The horse also had slight attacks of colic and yellowness of the mucous membranes.

Treatment. As the condition is always secondary, attention must be paid chiefly to the treatment of the primary disease.

5. Rupture of the Liver. Ruptura hepatis.

(*Hæmorrhagia s. Apoplexia hepatis.*)

Etiology. The comparative richness in blood and the softness of the liver tissue explain the rupture of even healthy livers when subjected to some sudden mechanical influence which causes a rise of internal pressure. Among such influences may be included jumping, falling, kicking, severe vomiting, etc. (Johne). Rupture of the liver may be caused by fractured ribs. In cattle foreign bodies passing through the stomach wall may also cause this accident. Parasites, such as the Pentastoma denticulatum in the sheep, fluke and Cystercercus tenuicollis in ruminants and pigs, have also been responsible.

As a predisposing cause of rupture of the liver, there is usually a degeneration of some kind of the liver tissue. In this connection, at least as regards the horse, amyloid disease is the most important. Parenchymatous degeneration and more frequently fatty degeneration, abnormal conditions of the blood vessels in acute infectious diseases such as anthrax, purpura, hemorrhagic septicemia, hepatic lesions of a glanderous or tuberculous nature, the latter especially in birds, abscesses (Schüler) and neoplasms (angioma, sarcoma, carcinoma) sometimes determine rupture of the liver. Elevations of blood pressure, such as occur in the early stages of inflammatory processes and in the veins of the liver as a result of diseases of the heart and lungs, are predisposing causes of hemorrhage of the liver. Hemorrhage may also follow embolism of the hepatic artery or portal vein. In all these instances the hemor-

rhage occurs either spontaneously or in response to some trifling external influence.

According to Wörz too liberal a diet may cause parenchymatous hemorrhages in the liver, the over-filled stomach and intestines causing pressure on the liver and portal vein and thus producing congestion of the vessels of the liver. As mentioned by Noyer, rupture is very likely to occur in such cases when the liver is amyloid.

Anatomical Changes. In cases where there is rupture of a large number of small vessels the liver is beset with small dark punctiform hemorrhages. Such hemorrhages and even large ones may later become enclosed in fibrous capsules. These sometimes become calcified (Bruckmüller), or, after absorption of the granular débris, converted into cysts with red-tinged serous contents. On the other hand, old hemorrhages may be indicated by fibrous cicatrices only.

In large hemorrhages, such as commonly occur in amyloid livers, there is extensive breaking down of the liver tissue, forming large cavities with soft irregular walls and containing coagulated blood (hematoma). In such cases the weight of the liver is increased. In a case reported by Trasbot the liver of a horse weighed 33 kilogrammes. When the superficial layers of the liver tissue are broken through, the blood collects under the serous covering and elevates it. In these cases, there is a cavity filled with blood, immediately under the serous membrane. By the bursting of such hematomata large quantities of blood may be set free into the peritoneum.

Symptoms. Small hemorrhages into the liver tissue cause no symptoms. If the hemorrhage is severe, symptoms of internal bleeding are produced. The animal suddenly becomes dull, it has an anxious expression, the pulse is accelerated and weak, the mucous membranes are pale and dry and the peripheral parts of the body are cold. Soon after, there is local or general sweating, muscular tremors and staggering follow, and finally the animal dies in from one to ten hours with convulsions.

In cases where the hemorrhage is not so profuse, the symptoms are less severe. In many cases the only abnormalities observed are weakness, acceleration of the pulse, inappetence, jaundice, slight constipation, stiffness of gait, susceptibility to pressure over the liver or extension of the area of hepatic dulness. The latter symptom has been observed by Weber in a number of cases in the horse. In one particular case in a horse, a hematoma in the neighborhood of the portal vein caused marked swelling of the spleen. In some such cases there is improvement, at least for a time, in others, there is a sudden wasting and death in five to six days.

In a case recorded by Dieckerhoff a horse lost so much blood that it was incapable of work for 3 or 4 months, but afterwards worked in a wagon for a year.

In the cases recorded by Wörz in which there were repeated hemorrhages, the horses at first showed temporary digestive disturbances and slight abdominal pain.

Later there was anemia, dullness, wasting, edema of the legs, under surface of the body and sheath. These symptoms were repeated with gradually increasing severity, the dullness and depression became very pronounced, the feces were dry and mixed with large quantities of imperfectly digested food and the urine was dark brown in color. Finally the animals died in a state of complete exhaustion, if a rupture of the liver did not supervene and kill them.

Treatment. When there is a suspicion of hemorrhage of the liver the most important factor in the treatment is absolute rest. Ergotin, extract of hydrastis or adrenalin may be used. The dose of the latter for a horse is 1 to 5 cc. of a 1:1000 solution per 100 kilograms body weight; and for dogs, 0.5 cc. The drugs are administered by subcutaneous injection. The constipation should be relieved by enemata, and where there is great weakness of the heart, camphor and caffein are indicated.

3. Acute Parenchymatous Hepatitis. Hepatitis parenchymatosa acuta.

Under the term acute parenchymatous hepatitis are included inflammatory diseases of the liver substance in which, in addition to cellular infiltration and hyperemia, there is pronounced cloudy swelling and fatty infiltration of the liver cells.

Etiology. Acute hepatitis is rarely observed as a primary condition. It is generally caused by poisonous substances or by bacteria which reach the liver by way of the portal vein from the intestine. Possibly the organisms sometimes reach the liver by way of the bile duct. Apart from those caused by defective food or poisonous plants, cases of hepatitis sometimes occur in association with catarrh of the stomach and intestine, but in which the liver disease is apparently primary. Two such cases are recorded by Fröhner in which there were no lesions of any importance in any of the other organs.

The two cases of fatal acute hepatitis described by Kas and Lipa in the horse were probably cases of amyloid disease. Robertson and others have stated that certain plants belonging to the genus Senecio have in some cases produced acute hepatitis associated with catarrh of the stomach and intestine, but that the lesion so caused is usually in the nature of a chronic interstitial hepatitis.

Gilruth and especially Theiler have shown experimentally that many species of Senecio (S. Jacobæa, S. latifolius, S. Burchelli) when fed daily in quantities of from 50 to 500 gm. will, at the end of 50 to 125 days, or exceptionally even sooner, and as long as 96 days after the discontinuation of feeding, produce in horses and cattle acute or chronic hepatitis accompanied by a light gastro-intestinal catarrh. This disease occurs under natural conditions and frequently in different regions of South Africa and Australia. It also occurs in swampy regions.

As a rule, acute hepatitis is purely secondary to some acute disease. It may be seen in gastro-enteritis, or it may be caused by certain poisons, lupinose, phosphorus, arsenic, etc. The symptoms are generally obscured by those of the primary disease and consequently the clinical aspect of the condition is of little importance. Not rarely acute hepatitis is set up by the migration of certain animal parasites (distomes, cysticerci, pen-

tastomes, sclerostomes). This type of hepatitis will be considered under a separate heading.

Anatomical Changes. The liver is enlarged and its edges are rounded. The cut surface is dull and the glandular arrangement is obscure, but small reddish patches or dark red hemorrhages may be present. In acute Senecio hepatitis (Senecio or ragwort poisoning) the liver is usually enlarged and firm. It usually shows the delineation of a nutmeg liver with distention of the central vein and a grayish or yellowish coloration of the edges of the lobules and sometimes a granite-like color. On microscopic examination the central veins and the more central portion of the capillaries are dilated, the parenchyma shows cloudy swelling, and, as a rule, different stages of fatty degeneration, vacuole formation, and atrophy of the liver cells in the adjoining trabeculæ. Besides round-cell infiltration of the lobules, frequently fibroblasts and proliferation of the bile ducts, myocarditis with fatty infiltration and fatty degeneration of the kidneys are also observed.

Symptoms. Susceptibility to pressure over the region of the liver, evidence of pain during defecation, and finally jaundice indicate acute hepatitis, provided the history of the case is in agreement with that. Diagnosis can be made earliest in the dog. Fröhner observed two cases in which, besides great weakness, there was severe jaundice and susceptibility to pressure over the liver. In one case there was fever.

In acute hepatitis caused by species of Senecio there are observed obstinate diarrhea, pronounced tenesmus, frequently prolapse of the rectum, excitement, and in 2 to 3 days a fatal termination. Theiler observed colic in two cases. Pronounced icterus is always present and is frequently accompanied by punctiform hemorrhages in the membrana nictitans. Acceleration of the pulse up to 100-120 per minute, pounding heart beats, dark brown discoloration of the urine, sometimes hemoglobinuria and albuminuria, regularly great depression, lowering of the head or supporting it on some object, pushing forward and a swaying gait were also observed by Theiler.

Treatment. The line of treatment indicated is the administration of neutral salts, various mild purgatives or disinfectants. The quantity of fat in the diet should be restricted, and the diet should be changed.

Acute Diffuse Parenchymatous Hepatitis of the Sheep. Azary records the annual occurrence during the months of January and February of a peculiar disease among the pregnant ewes on a farm in Hungary. During a period of five years the losses amounted to 473 ewes (29% of the flock). The disease broke out a few days after the animals were brought in, and attacked principally the ewes that were in the most advanced stage of pregnancy, and of these only the best nourished.

The symptoms presented were as follows: The animals became markedly dull and lay on the ground with half-closed eyes. On the second or third day, after a partial recovery, rumination was suspended and the animals again became semi-comatose. The temperature was normal at first, but then rose to 40°-41.5° C. before death. At the outset the pulse was accelerated and later became weak. Respiration was hurried throughout, and in the late stages was of the Cheyne-Stokes type. The animals lay on their sides seeking to relieve the pressure over the region of the liver. The urine was reduced in quantity and contained albumen, epithelial cells from the straight tubules of the kidneys and from the bladder, and leucin bodies. On the 5th or 6th day, there was complete apathy, grinding of the teeth and muscular tremors. The wool came out in large tufts and the animals died about the end of the first week in a state of complete exhaustion.

At the postmortem the liver was found to be enlarged, anemic, more or less soft in consistency, yellowish or exceptionally brownish-yellow in color, as in cases of poisoning by phosphorus. The lobules were grayish-yellow and translucent, with a grayish-white peripheral zone. The margins of the lobules were indistinct. Besides numerous droplets of fat the liver cells contained rounded yellow translucent bodies that were not soluble in alcohol or ether (leucine?). There was no evidence either of parenchymatous or fatty degeneration in the kidneys but the above-mentioned bodies were present in the lumen of the tubules. Other lesions were: hemorrhages of the serous membranes, and parenchymatous degeneration of the heart-muscle.

Azary described the disease as an acute diffuse parenchymatous hepatitis which showed considerable resemblance to the yellow atrophy of the liver seen in fashionable women during the last months of pregnancy. The cause of the condition could not be determined.

A similar disease was observed a year later by Czakó & Hartmann. In this case, there was no fever and the disease took a benign course. Flóris has recently recorded a disease occurring in Hungary, among ewes that had been kept indoors for weeks. He connected the disease with the number of fetuses in the uterus of the ewes, as in every case that came under his observation there were three.

A similar disease was described by Murillo & Izcara as an infectious inflammation of the bile-ducts and gall-bladder. This was referred to a bacterial invasion.

Haubold records a disease occurring among sheep during the autumn months, the symptoms of which were: loss of appetite,great weakness, increased thirst, jaundice, soft, pultaceous, and, at the last, blood-stained feces. The cause was probably the exclusive use of wet grass and clover, for no further symptoms were observed after the diet was altered.

According to Ömler, the following set of symptoms were seen either on the same or the following day, in a flock of sheep feeding off fields of good rye stubble; great weakness and somnolence, inappetence, watery diarrhea that was rather offensive and sometimes mixed with blood, staggering gait, pain on pressure over the abdomen, hurried respiration, fever, purulent discharge from the nose that was sometimes blood-stained, rather bright red coloration of the mucous membrane of the mouth. One-third to two-thirds of the animals died within 1 to 2 days. In addition to inflammatory changes in the stomachs and intestines, the liver was found to be grayish-yellow in color and easily torn.

Enzootic Hepatitis of Young Pigs. Semmer has recorded an enzootic among two-months-old pigs that caused great losses in Russia. In these cases the liver was enlarged and nodular. The cut surface presented a variegated mosaic-like appearance, dark red patches alternating with bright red or grayish-yellow areas. The peritoneum and sometimes the pleura contained a serous exudate, the intestines were inflamed, the urine contained albumen. Between the liver cells, which were infiltrated with fat, there were small-celled infiltrations and collections of extravasated red blood corpuscles. The only symptoms observed were debility and inappetence just before death. In the liver, spleen and blood, Nonewitsch found large cocci, cultures of which when inoculated into young pigs caused death in 7 to 8 weeks. At the postmortem the above-described lesions were found. From the long period elapsing between inoculation and death and from the fact that only pigs of about 2 to 4 months old were attacked, the author concludes that the infection occurs immediately after birth, possibly by way of the navel.

According to Kleinpaul, Brädel and Willerding the disease occurs in Eastern Prussia and it has recently broken out in a very virulent form. During the year 1906, the losses were greater than those due to swine plague. According to the histological investigations of Brädel, who describes the disease as hepatitis haemorrhagica mortificans, the condition is not due to cysticerci or distomes as supposed by Seiler and Wolffhügel. Brädel also points out that the hepatitis may be connected in some way with swine fever.

7. Acute Yellow Atrophy of the Liver. Atrophia hepatis flava.

Under this term are understood processes of severe parenchymatous and fatty degeneration associated with a rapid disintegration and atrophy of the liver cells which are directly due to toxins, or to a predisposition of the liver cells, and which result in disturbances of metabolism as well as in the detoxicating activity of the liver cells.

Etiology. In animals such a process in the liver is brought about especially by toxins foreign to the body, particularly phosphorus, more frequently, however, by lupines and by other unknown vegetable toxins. Particularly in cases which Theiler sometimes observed in large numbers of horses in South Africa, a rapidly progressing disintegration of the liver cells was brought about by toxins of forage plants acting similarly to Senecio toxin; the liver was sensitized by immunization against horse sickness and the simultaneous presence of infectious anemia, or by exhausting work or other influences.

According to Leonpacher, a disease in cattle similar to acute lupinosis occurs in the vicinity of New Orleans, Louisiana, and is produced in winter by a bean-like forage plant, Mucuna utilis (velvet bean), belonging to the Leguminosæ, if after a long rainy spell the cattle are pastured on fields from which all the velvet beans have not been harvested. Beans that have fallen to the ground and have germinated in the mud are particularly dangerous. (The bean is also toxic for man and chickens.)

According to Rickmann, geelziekte in sheep (yellow disease) in Southwest Africa is produced by the toxin of a leguminous plant having a creeping stem, yellowish flowers, and pods 5 cm. long. The disease exhibits symptoms similar to lupinosis and terminates fatally in 1 to 2 days. Callot observed multiple cases of an unknown origin in Uruguay. Unknown toxins were probably also responsible for cases observed following the ingestion of soured potato mashes (Haubner), pea or vetch straw (Reinemann, Jansen) and green vetch (Stoehr). Intoxication by spoiled feed (hay from inundated regions, according to Haubner and Franze), as well as the toxic effect upon the liver tissue in certain types of gastroenteritis and septicemia, must also be considered. Acute yellow atrophy of the liver occurs much more frequently than the literature would indicate, because, according to Joest, it is frequently diagnosed as acute parenchymatous hepatitis. Particularly is this true of the cases described as acute diffuse parenchymatous hepatitis of sheep and enzootic hepatitis of pigs.

Anatomical Changes. The liver is smaller, soft, and of a light to okra-yellow color. On section there are found somewhat firmer, red areas with far advanced atrophy of the liver cells, of a mosaic design (according to Joest, especially distinct in the dog). The still intact liver cells show advanced fatty degeneration. At the same time different degrees of cellular infiltration, degeneration of the epithelium of the bile ducts, and proliferation of the smallest bile capillaries are found. Fatty degeneration of the kidneys is also found, and sometimes acute swelling of the lymph glands, tumefied spleen, gastrointestinal catarrh, and hemorrhages in the different organs. Acute yellow atrophy in horses, as observed in South Africa, shows a certain peculiarity in that the necrotic process begins in the center. Accordingly, the edges of the lobuli are relatively less changed, while the central portion and frequently even the whole lobule is changed into a homogeneous mass. Besides, the myocardium shows pronounced parenchymatous and fatty degeneration and the endocardium and pericardium are studded with hemorrhages.

Symptoms. The onset of the disease is marked by sudden depression and weakness, fever, jaundice of the mucous membranes, inappetence and, in some cases, attacks of colic with tenderness of the abdomen (Zündel). In the later stages there is diarrhea. The jaundice and dullness soon become very marked, but at intervals there are symptoms of excitement.

As a result of the coincident disease of the kidneys, the urine is diminished in amount, reddish-brown in color and contains bile pigments and albumin. In the human subject leucin and tyrosin are sometimes present in the urine. On the addition

of acetic acid these are precipitated in the form of rounded masses and needlelike crystals.

Death usually takes place in about six or eight days. The reduction in the amount of urine and the presence of albumin in it are unfavorable signs.

In acute atrophy of the liver in horses in **South Africa**, Theiler observed, in cases with a less severe course, depression with the head lowered, long-continued walking and leaning against the wall of the stall (evidenced by lime spots on the projecting parts of the body in whitewashed stalls, skinned places) *and long-continued standing with the head resting against the* wall or other objects. In cases with a severe course there is rapidly increasing excitement until the animal is almost furious and tries to run away, running into and over obstacles that may be in its way; in the stall there is constant walking with the head held in an unnatural position; severe bodily injuries; rarely there is twitching or convulsion of the muscles, whereupon the animal drops into a profound coma. Icterus is often observed before the nervous disturbances make their appearance, while an accelerated and weak pulse appears somewhat later. Hemoglobinuria is also sometimes observed, albuminuria somewhat more frequently. Aminoacids (especially tyrosin) may be present in the urine (in one case up to 11% of total nitrogen). The disease runs its course in 24 hours, sometimes in 6 to 12 hours, or again in 2 to 3 days. Fever is not present.

French authors (Cruzel, Beauvais, Delage), have described an acute hepatitis in cattle in which the liver is enlarged and clay-colored. The symptoms are general malaise, inappetence, salivation, grinding of the teeth, staggering gait, jaundice, pain in the right side of the abdomen and constipation followed by diarrhea.

Treatment. Disinfectants, mild purgatives and diuretics may be tried, but so far all treatment has proved useless.

8. Lupine Disease. Lupinosis.

By this name is designated an acute parenchymatous hepatitis or an acute yellow atrophy of the liver caused by lupines and which also exhibits other symptoms of intoxication and in case of a protracted course terminates in cirrhosis of the liver.

Etiology. In certain districts in Northern Germany where lupine (Lupinus flavus, cœruleus and albus) is used as fodder, on account of its high percentage of protein, extensive outbreaks of disease sometimes occur among sheep, which are no doubt due to poisoning by these plants. In these districts the lupines are harmful only in certain years and in any year the plants may be poisonous on certain fields only. They are seldom poisonous in the fresh state, but only after a period of storage. Lupine never

causes disease when it has been exposed to rain or frost. The yellow lupine is the most dangerous, and the seeds, pods, leaves and straw are equally toxic. Goats, oxen and horses are also attacked. Cases are rare in horses, as they do not like lupine on account of its bitter taste.

(Attention has already been called to the fact that in North America under certain conditions a disease similar to lupinosis is caused by **Mucuna utilis** [velvet bean] [see page 441].)

The investigations of Arnold & Lemke, Liebscher, Kühn and Roloff, regarding the poisonous substance contained in lupine are well known. It may be extracted from the plant by an aqueous alkaline solution, such as 2% soda. The extract causes the same symptoms as the plant itself, and the plant is non-toxic after extraction. The toxic substance has received a variety of names. Kühn calls it "Ictrogen," and Arnold and Schneidemühl, "Lupinotoxin." It is with difficulty soluble in water, insoluble in glycerine, alcohol and ether. It is not destroyed by a temperature of 190° C. dry heat even after three hours but by steam at 2½ atmospheres pressure its pathogenic power is lost.

The circumstances which control the production of lupinotoxin are not yet known, but it cannot be denied positively that the toxin is produced by fungi which grow on dead plants or on the food itself after storage, and that it is allied to the bacterial and fungoid toxins. This view is supported by the facts that the toxicity is retained in food that has been left in the open for a long time, and also it would be quite impossible to explain why lupine grown on neighboring land fed at different times and in different manners sometimes shows great variation in toxicity.

Arnold and Schneidemühl's method of extracting the toxin: Finely ground lupine is mixed with 1½% soda solution to produce a thin pulp. This is maintained at 40 to 50° C. for two days and then subjected to pressure. The liquid so obtained is placed on a water bath at 60° C. and after the addition of acetic acid is filtered. Lead acetate is added and sulphuretted hydrogen passed. The filtrate is evaporated at 50° C. and the semi-liquid residue is mixed with 15 volumes of alcohol. The precipitate which falls in about 24 hours forms when dried a brown shining resinous mass. Five grams of this produce typical symptoms in a dog. After further purification the material is found to be of an albuminous nature.

Liebscher has isolated and experimentally investigated the alkaloids contained in lupine and has shown that they always produce paralysis of the medulla oblongata, and further, that absorption from the stomach is very difficult and that no causal connection can be established between them and lupinosis.

Pathogenesis. Lupinotoxin principally affects the mucous membrane of the alimentary canal and after absorption affects the liver, setting up fatty degeneration and catarrh of the gall bladder. The toxin circulating in the blood causes parenchymatous degeneration and fatty degeneration of all the solid organs.

Anatomical Changes. In very acute cases there is, in the early stages, cloudy swelling of the liver and jaundice, either of the liver only or of the whole body. Later, fatty degeneration occurs, the liver appearing soft, easily torn and of an intense yellow color. There may be a number of red foci. There is a

simultaneous parenchymatous degeneration or fatty degeneration of the kidneys, heart muscle, and certain groups of muscles, while there is severe inflammation associated with small hemorrhages in the abomasum, ileum and large intestine. There are numerous hemorrhages in the serous membranes, skin and subcutaneous connective tissue, and also an edematous infiltration.

In chronic poisoning the liver shows chronic interstitial inflammation, and not rarely a nodular condition produced by shrinkage. These lesions are associated with the presence of fluids in the body cavities, enlargement of the spleen and chronic nephritis.

Symptoms. In sheep the first symptom is lack of appetite, especially for lupine, other foods being eaten for some time longer. At almost the same time symptoms of cerebral excitement or great depression occur. There is great weakness. The animals lie down a great deal or stand with their heads hanging, making vague chewing movements, grinding their teeth, swaying from side to side until they drop quite unconscious.

According to Roloff the temperature is elevated at the onset of the disease but it shows pronounced irregularity, and towards the end there is a marked fall. Both pulse and respiration are greatly accelerated in the later stages.

In the majority of cases there is jaundice of the visible mucous membranes, especially in animals that are most severely attacked. At the commencement there is constipation and the dung is hard and covered with yellow mucus. Later there is diarrhea, the feces being mixed with blood. The urine is passed at short intervals, it is dark red in color and contains large quantities of albumin, bile pigments and bile acids. In the sediment there are different kinds of epithelial cells and renal casts.

In occasional cases there is an erysipelatous swelling of the skin of the head, especially in the region of the ears and nose. A serous yellow liquid exudes from the surface which dries up and forms crusts (Stöhr). Exceptionally there is a purulent or sanguinolent discharge from the nose (Roloff).

In the horse the symptoms presented are more or less like those in the sheep, especially with regard to the refusal of food and the intense depression and jaundice, which are the most prominent symptoms. There may also be a yellow mucous discharge from the nose, erysipelatous inflammation of the skin of the nostrils and lips, which may become gangrenous, and inflammatory edema of the limbs.

Course. In acute cases death occurs in 3 to 5 days, but in the majority of cases it is delayed until the 9th or 11th day. If the food be changed immediately when the symptoms appear, improvement sets in about the 5th or 6th day, and there is rapid and complete recovery. In a proportion of cases the recovery

's not complete and the animals die later, showing symptoms of chronic hepatitis and general cachexia.

If lupines that are toxic to only a slight degree be used for food over a long period, symptoms of chronic lupinosis develop, the animals showing progressive anemia and cachexia. Jaundice is absent or only slight, but inflammation of the skin is sometimes observed.

Treatment. In the absence of an antidote efforts must be directed towards preventing the absorption of the toxin by giving acid solutions and removing the toxin from the intestine as rapidly as possible by means of oil purgatives (Roloff).

If the lupine has been proved to be poisonous, its use must be discontinued for a time. The food can be rendered harmless by exposing it to rain in small heaps and using only the superficial layers for feeding purposes, or it may be soaked in 1% soda solution, with renewal of the liquid every two days, and then dried, or by submitting it to steam at a pressure of two atmospheres. Slightly poisonous lupine may be mixed with good food in the proportion of 1:6-10 without danger. Finally it is advisable to test every fresh supply of lupine upon a few sheep.

9. Suppurative Hepatitis. Hepatitis suppurativa.

(*Embolic hepatitis.*)

Etiology. Suppurative hepatitis is usually a metastatic lesion. The pyogenic bacteria, responsible for its production (streptococci and staphylococci), reach the liver either by way of the arterial blood or by way of the portal vein. In cases where the infection occurs by way of the portal vein, the source of the infection is gastro-enteritis, and especially when there is ulceration or necrosis of the mucous membrane (enterogenous abscess of the liver [Joest]). Other causes are suppuration of the spleen, intestinal wall or mesenteric glands and, finally, disintegration of thrombi in the umbilical vein in new-born animals. According to Schumann, the abscesses in the livers of calves are associated with suppurative inflammation of the navel (omphalogenous abscess of the liver [Joest]). Pyogenic bacteria may gain access to the liver by way of the blood stream in any severe inflammatory condition, but especially in strangles, ulcerative endocarditis, and gangrenous pneumonia. Infection may occur by way of the bile ducts, owing to necrosis and inflammation of the walls, caused by gallstones, foreign bodies (heads of grain, sand) or animal parasites, the infection then spreading to the liver tissue.

Injuries to the liver are most common in cattle and are usually due to the penetration of foreign bodies from the stomach. Injuries from the outside, such as horning, may have the same results. Pyogenic bacteria may be introduced into the

liver in this way or the liver tissue may be broken down, and any bacteria circulating in the blood may localize there.

Finally, suppuration is frequently caused by echinococcus cysts, tuberculosis and actinomycosis. In these cases the primary condition is of special importance. In nodular necrosis of the liver there is sometimes liquefaction of the necrosed tissue, leading to the production of abscess-like lesions, which differ from the true liver abscesses in that they contain only structureless débris.

At the Budapest slaughter house during a period of 5 years suppurative hepatitis was found in 0.2% of slaughtered cattle and in 0.09% of calves. Lisi found numerous abscesses in the livers of lambs.

In the tropics abscesses in the liver are of very common occurrence and are very often associated with enteritis. The disease has been observed by Smith in India and by Griffault in the Sudan.

Anatomical Changes. The liver contains numerous abscesses, varying in size up to a nut. Their contents may be creamy or dry and crumbling. The color varies from yellow or reddish to green. Occasionally the pus has an offensive odor. In suppurative hepatitis of umbilical origin in calves, the abscesses are principally found in the left lobe, on account of the distribution of the portal vein (Schumann). In recent cases the liver tissue is in a condition of parenchymatous or fatty degeneration, but in cases of longer standing the abscesses are provided with walls of fibrous tissue and the surrounding liver tissue is traversed by strands of fibrous tissue. In other words, the lesion is one of chronic interstitial hepatitis. The superficial abscesses project above the level of the liver tissue and fluctuation can be felt. The serous membrane of the liver may be covered with fibrin or thickened.

For various reasons the abscesses are usually few in number, and when due to an injury there is, as a rule, only one. In such cases the abscesses may attain an enormous size. Felisch reports one in which the abscess measured 20 cm. in its greatest diameter. Abscesses of this size project above the surface of the liver and are usually adherent to the diaphragm, or possibly to the stomach or large intestine. In rare cases rupture of such an abscess causes a suddenly fatal peritonitis.

In the liver abscesses of an ox Grips found the bacillus pyogenes bovis. Künnemann repeatedly found the necrosis bacillus and Lisi found staphylococci in the abscesses in the livers of lambs. According to Schumann omphalogenous abscesses in calves are caused by various bacteria but *especially* by streptococci and staphylococci. In other cases he found colon bacilli and the bacillus pyocyaneus. The abscess-like *necrotic foci* are caused by the necrosis bacillus. (Bürgi has described a purulent hepatitis of the hare that is due to a staphylococcus.)

According to Stubbe an abscess on the anterior surface of the liver sometimes elevates Glisson's capsule and becomes adherent to the diaphragm, the extent of the adhesion gradually extending until the abscess wall loses its connection with the liver and remains attached to the diaphragm, the convexity of the liver showing a depression corresponding to the position of the abscess.

Symptoms. Single and, occasionally, numerous small a scesses may produce no noticeable symptoms, or at most, certain amount of wasting. In other cases suppurative hepatil is a complication of another disease, and thus the symptoms which it gives rise are completely obscured. It is only in ra cases that a moderately rapid process of abscess formation the liver causes visible symptoms. In addition to disturbanc of digestion, there may be persistent fever of no particular typ or there may be simply occasional elevations of temperatur An increase in the amount of indican in the urine is demo strable. These symptoms are indicative of some internal su purative process; the more or less pronounced jaundice, and tl presence of bile pigments in the urine point to suppurative hep titis (Smith).

In the dog and ruminant enlargement of the area of hepat dullness has some diagnostic importance, as has also swellin in the hypochondriac region in the former. In all animals ther is pain on pressure over the hepatic area. According to Moui leron and Chauffart, hepatic dulness can be discovered in th horse in some cases. In cattle there may be symptoms of trau matic gastritis (indigestion, shallow respiration and groanin during motion). According to Griffault, the following symp toms may be seen in the horse: Susceptibility to pressure ove the hepatic area, pain during defecation, coughing and straining marked fullness of the veins of the abdominal wall, edematou swellings and irregular temperature. The enlarged liver may b palpated on the right side behind the costal arch. Albrecht ob served coughing in the horse.

In order to ascertain the position of the liver-abscess Griffault advises punc ture in the 16th or 17th intercostal space close under the costal arch. The operatio is not without danger as infection of the peritoneum is likely to occur if the absces is superficially placed.

Treatment. Internal treatment is utterly useless. If the diagnosis be sufficiently certain, surgical intervention may be attempted.

Griffault operated as follows: An incision was made through the abdominal wall as far as the peritoneum, immediately below the costal arch on the right side. The peritoneum was then approached to Glisson's capsule so that its inne surface was in contact with the abscess wall. The abscess cavity was drained and washed out and a bandage applied. In this way two animals were cured.

10. Nodular Necrosis of the Liver. Necrosis nodosa multiplex hepatis.

Nodular necrosis of the liver is characterized by the presence of tumor-like, dry nodules in the liver substance, which undergo a process of gradual softening from the periphery.

Occurrence. The disease is of very frequent occurrence in cattle. Cases in the sheep are more rare, and in the horse, pig

and dog quite exceptional. In the sheep it sometimes occurs as an enzootic (Berndt).

Etiology. As shown by the investigations of McFadyean, Bang, Schütz, Kitt and Meyer, the cause of the condition is the *necrosis bacillus* (Bang).

In natural cases the liver becomes infected by way of the blood stream. In most cases the bacillus reaches the portal blood from the intestine. It is not essential that any lesion should be produced in the mucous membrane. In new-born calves infection may take place by way of the umbilical vein. In 15 cows examined by Berndt, either just before or just after calving, the bacillus could be found in the diseased uterus. In certain cases a connection can be established between the disease and the use of very dusty or mouldy food, or the stabling of the animals in dirty stables.

Eisenmann found necrotic foci in the liver associated with chronic inflammation in some cases of swine erysipelas. Caseous foci are sometimes found in the liver of sheep and calves due to pseudo-tubercle bacilli. These foci bear considerable resemblance to necrotic lesions in the process of softening.

Pathogenesis. The necrosis bacillus is generally arrested in the smallest branches of the portal vein, or occasionally in the hepatic artery. Multiplication takes place there and the bacilli pass through the vessel wall and penetrate into the surrounding liver tissue. The center of the lesion gradually becomes necrotic and the bacilli are to be found in the peripheral parts arranged in radiating bundles of filaments. An inflammatory reaction sets in around the necrotic area, produced in part by the products of tissue destruction and bacterial toxin and in part by the necrotic tissue acting as a foreign body (McFadyean).

Anatomical Changes. The liver, which may be more or less enlarged, is either normal or slightly yellowish in color. The surface of the organ shows sharply defined rounded protuberances of a light brown or yellow color. These are of a firmer consistency than the surrounding tissue. The capsule of the liver covering the superficially placed nodules is thickened and covered with a thick layer of fibrin. Some of the superficial nodules may be eroded and covered with a purulent liquid. There is often secondary sero-fibrinous peritonitis, which is most marked in the neighborhood of the liver.

When the disease has been in existence for a long period the nodules comprise a dry necrotic center, a zone in which the inflammatory reaction has produced softening and an external capsule of fibrous tissue. Finally the central part becomes converted into a viscous yellow material resembling pus, and is surrounded by a fibrous capsule which may measure 3 mm. in thickness.

In cases of chronic swine erysipelas Eisenmann found the liver reduced in size and beset with white or yellowish centers.

Symptoms. According to Berndt the symptoms are inappetence, high fever, weakness and painfulness of the liver. The animals move with difficulty, respiration is accelerated and shallow, pressure over the liver causes pain. After three days the animals become very weak and lie down continuously. Respiration is very rapid and difficult, there is constipation followed by diarrhea, the abdomen is painful, the temperature falls, jaundice sets in and the animals soon die. Otto saw two cases in which there were symptoms resembling those of parturient paralysis after the disease had been in existence for 1 to 1½ weeks.

In cases where the number of lesions is small, or where subsequent processes are less rapid, the symptoms are not severe or there may be no evidence of any disturbance of health.

In a stable enzootic of necrobacillosis (Necrobacillosis bovum epidemica) observed by Miessner & Bartels, the disease was manifested in the individual animals as a catarrhal pneumonia, enteritis and hepatitis, catarrhal inflammation of the udder with necrosis of the skin, panaritium and abortion. During the prodromal stage only diminished appetite, loss of condition, and cough were observed; but during the open stage of the disease there was high fever and the animals rapidly lost in strength. At the commencement of the enzootic only cases of bronchopneumonia were seen; after four weeks digestive troubles were more apparent and after four more weeks cases of panaritium. The duration of the disease was several weeks. Recovery did not occur.

Diagnosis. If no suppurative process can be discovered in any other part of the body and if the cow be in the last stages of pregnancy, or just calved, the symptoms already described (pain on pressure over the region of the liver, jaundice, and later evidence of peritonitis), justify a suspicion that the disease is in existence. It is, however, difficult to arrive at a certain diagnosis or to exclude the possibility of purulent or pseudotuberculous hepatitis.

Treatment. Treatment has so far proved useless and the immediate slaughter of the animal is indicated.

11. Chronic Interstitial Hepatitis. Hepatitis interstitialis diffusa chronica.

(*Cirrhosis hepatis.*)

This condition consists essentially in a proliferation of the interstitial tissue at the expense of the liver substance. In some cases the new fibrous tissue does not contract, either for a long time or at all, and atrophy of the liver substance is seen only at places (hepatitis indurativa hyperplastica). In other cases there is an early shrinkage of the connective tissue and a consequent reduction in size of the liver and destruction of liver parenchyma (cirrhosis atrophica hepatitis).

Occurrence. In certain districts the disease is of frequent occurrence and not rarely there are outbreaks, causing great

losses, since, sooner or later it has a fatal termination. Large numbers of cases are observed under *certain conditions*. Besides these, large numbers of cases are due to the migrations of animal parasites, especially in pigs, sheep and oxen. Otherwise, the disease is sporadic in all animals.

The *form of chronic hepatitis known as* "Schweinsberg Disease" was first observed in Schweinsberg, Ohmtal. Since then it has been seen in some districts in Bavaria and the Rhine provinces. The disease varies in severity from year to year and attacks freshly imported horses more frequently than native ones. In South Africa the disease occurs among horses and cattle, both sporadically and as an epizootic (Robertson). In New Zealand it is known under the name "Winton's Disease" (Gilruth), in Nova Scotia as "Picton Cattle Disease" (Wyath-Johnston), and in South Dakota as "Bottom Disease" (Schröder, Smith).

Etiology. There is no doubt that the long continued administration of poisonous materials produces primary chronic hepatitis. *In this connection plants used for food call for special* consideration. These plants in swampy districts contain some poisonous material, the nature of which is not yet known. The socalled "Schweinsberg Disease" was seen only in horses that had grazed on lands which are often flooded, or that had received food grown on such land, while the farms on higher ground in the same district never had cases of the disease or suffered only to a slight extent. The plants grown in these districts contain some substance that is irritating to the liver tissue. Schlegel and Adelmann have recently shown that the disease may be no more than a generalized sclerostomiasis, and that the *chronic inflammatory changes in the liver are produced by the* migrations of the larvæ of sclerostomes.

The idea that *certain plants contain substances capable* of causing chronic hepatitis does not agree with the experience obtained with regard to chronic lupinosis. However, the disease has been observed after long continued feeding with several plants of the Senecio group (S. Jacobea, S. latifolia, S. Burchelli) in New Zealand, North America and South America (Smith, Gilruth, Robertson, Wyath-Johnston, Schröder), and it has been set up experimentally in cattle and horses by Gilruth and Robertson. Guittard has observed chronic hepatitis in geese fat*tened on maize.*

Gilruth fed two six-months-old calves on six pounds of S. Jacobea daily. Both calves died at the end of four weeks. To the naked eye their livers appeared unaltered but under the microscope there could be detected a perivascular connective tissue, slight thickening of the capsule and commencing destruction of the liver cells.

Robertson experimented with calves, adult bovines and horses, and found that the administration of large quantities of S. Burchelli and latifolia caused gastro enteritis and venous hyperemia of the liver in a few days, while small quantities fed over a period of weeks caused atrophic cirrhosis of the liver.

Since chronic hepatitis is frequently met with in people addicted to alcohol, the idea suggests itself that alcohol may be the cause of the disease in animals, in as much as it is present in the swill tub. One must not lose sight of the effect of fer-

mentation products that may be formed in this food during storage; and especially, as it has been shown by v. Baumgarten and Hansemann to be impossible to produce chronic hepatitis in experimental animals by the systematic introduction of alcohol into their bodies, either by subcutaneous injection or by ingestion.

In certain cases the disease is set up by chemical substances contained in rotten or mouldy food, or by certain digestive disorders of the alimentary canal itself. Of 350 pigs killed, belonging to innkeepers and brewers, Tschauner found 13 affected, but out of 5,700 farm pigs killed at the same time, only 3 were found similarly diseased. The former had been fed on the waste (potato peelings, etc.). Possibly the small percentage of alcohol present among the fermentation products played some part in the production of the disease.

In other cases cirrhosis of the liver is caused by other toxins having an endogenous origin (autointoxication); particularly in certain cases of indigestion, toxic products are formed in the intestinal tract (dyspeptic cirrhosis of the liver). In a case reported by Begeng in a cow the cirrhosis of the hypertrophied liver could be ascribed to a chronic gastrointestinal catarrh and could be traced to toxins carried to the liver from the intestinal tract with the blood of the portal vein. Krawkow and Lissauer produced cirrhosis of the liver in experimental animals by feeding a broth prepared from putrid horse flesh, while Boix obtained positive results in 6 to 8 weeks by the subcutaneous injection of monobasic fatty acids which are formed by abnormal fermentation in the gastrointestinal canal. Every radical change in nutrition may result similarly, as is indicated by the experiments of Chalatow with the yellow of eggs and brains of cattle, and those of Martin and Pettit with milk powder in rabbits.

Disease viruses are probably common causes of chronic hepatitis. Joest produced a condition resembling the Schweinsberg disease, both clinically and as regards the lesions by long continued injections of the bacillus suisepticus. Pronounced lesions of chronic hepatitis were observed by Eisenmann in several cases of swine erysipelas, and similar lesions may be sequels to infectious diseases (post-infectious chronic hepatitis).

Dantschakowa produced chronic induration of the liver in rabbits by repeated subcutaneous inoculations of the staphylococcus pyogenes aureus at intervals of 4 days in 7 to 15 weeks. This was followed by a localized small-celled infiltration and the development of a collagenic tissue in the center of the lesions and peripheral extension of the infiltration. A light form of cirrhosis of the liver is also observed in serum animals.

The disease is very frequently seen as a secondary condition. It is caused principally by animal parasites (fluke, Cysticercus tenuicollis, and the larvæ of sclerostomes). The sheep and pig are affected chiefly, but it is also met with in calves,

horses and rabbits. As already mentioned, Schlegel and Adelmann look upon the Schweinsberg disease as a generalized sclerostomiasis. The formation of fibrous tissue is due partly to the destruction of liver tissue and partly to toxic materials elaborated by the parasites themselves, particularly the fluke.

Chronic hepatitis may be caused through chronic inflammation of the walls of the bile ducts arresting the flow of bile, and by extension of the inflammatory process from the inter- and intra-lobular bile ducts to the interstitial tissue, the amount of connective tissue being increased. Engorgement with bile may lead to impairment of the nutrition of the epithelium of the bile ducts, and this may be followed by a bacterial invasion and so cause a production of fibrous tissue in the surrounding liver tissue, or the tissue production may be due to the ill-effects of the biliary engorgement on the liver cells themselves.

Purulent foci and tuberculous lesions may cause not only cirrhosis in their immediate neighborhood, but a diffuse lesion throughout the liver.

Chronic venous congestion caused by certain diseases of the heart and lungs, or by pressure on the posterior vena cava, may cause not only dilatation of the hepatic vessels (nutmeg liver) and an atrophy of the liver cells, but also an increase in the amount of connective tissue. Chronic hepatitis may also be seen in cases of thrombosis of the portal vein, which may either be due to the derangement of the liver parenchyma or may be the result of the cirrhosis, which has some previous cause.

Pathogenesis. If the noxious substances reach the liver via the portal vein, then the process will begin near the terminal branches of the blood vessels or the edges of the lobuli, while toxins and microorganisms reaching the liver with the arterial *blood will exert their injurious effect not only upon the margins* of the lobuli (in the region of the angles) but also upon the interlobular tissue, as will also those injurious substances contained in the bile (cirrhosis biliaris s. cholangioitica). Toxic substances which penetrate the capsule of the liver and are then carried along the lymph stream exert their injurious effect upon the subserous and interlobular tissue. Migrating parasites produce lesions quite independent of the anatomical structure of the liver (local inflammatory foci), while their toxins are brought into contact with other parts of the liver by means of the lymph stream. According to the nature of the injury and the affinity of the liver substance, there will be produced either a primary degeneration of the parenchyma of the liver, followed by proliferation of the connective tissue and of the bile ducts, or a primary irritation of the connective tissue with proliferation of the same and a secondary atrophy of the parenchyma of the liver and shrinking of the organ. The process often begins around several lobuli or groups of lobuli (cirrhosis

annularis), or in the form of islands (cirrhosis insularis), or again quite irregularly (cirrhosis lobaris).

In case of occlusion by pressure of numerous branches and capillaries of the portal vein in many cases of cirrhosis of the liver, stasis will follow in the region of the portal vein (corresponding to the decrease in the total cross section of the hepatitic circulation), thus increasing the portal blood pressure, which is still more increased by the compression of the open capillaries following atrophy of the lobuli and probably also by an increased inflow of blood from the anastomosing branches of the hepatic artery (Eppinger). On the other hand, the occlusion by pressure of numerous bile ducts, particularly in certain types of biliary cirrhosis, leads to bile stasis. Furthermore, the atrophy of the liver cells is also quite significant, because when pronounced it leads to disturbances in the secretion of the bile and also to disturbances in the metabolism of the body (carbohydrate and protein metabolism). Corresponding to the decrease in the capacity of the liver to neutralize toxins, it may also lead to autointoxication, which may sometimes produce death of the animal even before a pronounced stasis in the region of the portal vein has had time to develop. Partly by reason of the pressure exerted by the greatly enlarged liver upon the stomach, but mainly by the interference with the circulation of the gastrointestinal tract and of the pancreas, light digestive disturbances, with a simultaneous decrease in the absorptive capacity of the intestines, are produced. According to Theiler, in certain types of cirrhosis of the liver (dunsickness) a paralysis of the parasympathetic nerve fibers embedded in the vagus nerve and produced by the toxic action of the substances takes part in producing a dilatation of the stomach which occurs in such cases. In cases of focal distribution of the cirrhotic changes the retroactive effect of the cirrhosis upon the circulation in the portal vein, upon the digestion and upon metabolism may be entirely absent.

Anatomical Changes. In cases where the connective tissue has contracted, the liver has a tough and, in the later stages, a leather-like consistency. When cut into the tissue grates under the knife. At the outset there may be no visible reduction in the size of the organ and, in fact, it may be a little enlarged. In the later stages the reduction becomes more and more marked, until the liver may be only one half the normal weight. The surface appears irregular and granular. In some cases the surface is nodular. The serous covering may be thickened at places and the edges may be formed simply of a double fold of peritoneum. On the cut surface the compressed lobules stand out like small granules against the pale red or grayish-white connective tissue, which forms a wide network around either groups of lobules or individual lobules. These granules

appear of a faint or deep yellow color, with a tinge of green, on account of the fat and pigments contained in the liver cells. At a distance the whole organ looks yellow.

According to Kitt the liver in cases of Schweinsberg disease is at first enlarged, its surface uneven and of a reddish-brown or gray color resembling porphyry or granite. With the development and shrinking of the connective tissue the condition known as "granular atrophy" is produced.

The peculiar atrophy of the right lobe of the liver in old horses manifests itself in a thickening of the capsule of the liver and changing of the parenchyma of the liver into a sclerotic mass, whereby the affected lobe is reduced to a thin tongue-like appendage of the other two unaltered lobes (Barrier, Blanc, Garnier). According to Blanc, the atrophy is due to an insufficient supply of blood to the right lobe, since the arterial branch supplying this lobe turns off almost at right angles, while Garnier believes it is due to the action of infective material upon the capsule of the liver from the wall of the adjacent large intestine.

Nodular induration of the liver (hepatitis interstitialis chronica multiplex) occurs in chronic anthrax in hogs (Zwick, Schlegel, Bellut), following invasion of animal parasites (Joest and Felber, Trattner, Bellut), and, according to Bellut, also from toxic material in the blood. As a rule the liver is of normal size, but there will be found more or less numerous, sometimes almost innumerable, round, grayish-white nodules from the size of a dime to that of a quarter, with a reticulate structure and a similar boundary, which, below the surface, have the shape of a sphere or a hemisphere. Such nodules are sometimes depressed below the surface of the liver and may also partly coalesce. The parasitic nodules not infrequently contain a small cavity filled with necrotic material and a well-preserved parasite or its remnants, but frequently also only a pronounced eosinophilia as an indication of their parasitic nature (Joest and Felber). A pronounced eosinophilia is also found in the nodules, which Bellut believes to be due to intoxication, but is insignificant in the nodules due to anthrax. In these the reticulate structure is less pronounced toward the periphery and anthrax bacilli pathogenic for animals can be isolated from them.

In other forms of chronic interstitial hepatitis the liver appears enlarged. The outer surface and the cut surface appear smooth, or at most somewhat granular and deep yellow or greenish-yellow in color (cirrhosis hypertrophica s. hyperplastica). Livers so affected may weigh as much as 20 kilos in the horse and 20-25 kilos in the ox. Adam observed one case in an ox in which the liver weighed 15.0 kilos (Kitt). In the dog cirrhosis of the liver is frequently associated with fatty degeneration.

Evidence of congestion of the portal area and jaundice com-

plete the list of lesions. In Schweinsberg disease, pronounced catarrhal gastritis is a constant lesion.

A congenital hypertrophy of the liver has been frequently observed (first reported by Morot) in young calves in the first two weeks of life, which was called "giant liver of calves" by Ruppert, and "hyperplastic fibrosis of the liver" by Pick, and which Stenstroem interpreted as a hypertrophic or biliary cirrhosis. Joest, however, has shown that it is not a true cirrhosis but is merely a congenital gigantic development, particularly of the periportal connective tissue, resulting solely from a disturbance of development of the tissue. Such a liver may be enlarged up to 3 to 6 or even 12 kg., has rounded edges, a smooth surface, a firm consistency, and is light gray in color; or again, the surface may be lightly nodular due to the presence of

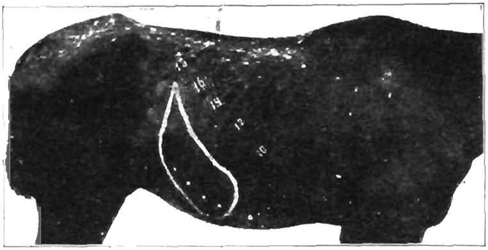


Fig. 80. Enlargement of the area of hepatic dulness in chronic hepatitis in the horse. The area enclosed by the line indicates the area of dulness, the white dots the costal arch, and the numbers indicate the position of the corresponding ribs. The liver in this case weighed 19.7 kilos.

numerous whitish-gray nodules and cysts up to the size of a pea and containing a colorless fluid. Other than frequent icterus, the anomaly does not produce any disturbances of health, and such calves are frequently fattened for slaughter.

Symptoms. The onset of the disease is unnoticed as a rule and even in later stages the symptoms are frequently only those of an indefinite digestive disorder. Capricious appetite, gaping and vomiting (observed by Moens in the horse) indicate severe gastric catarrh. There may be either constipation or diarrhea. Horses show symptoms of colic after eating large quantities of coarse food (especially straw mixed with dung) causing dilatation of the stomach (Imminger). In cases in cattle, caused by plants of the genus Senecio, there is severe diarrhea, tenesmus and often in consequence prolapse of the rec-

tum. Horses show symptoms of "sleepy staggers." Persistence of these symptoms causes the animal to lose condition. The mucous membranes appear pale and sometimes yellow. In Schweinsberg disease there may be early congestion of the mucous membranes. The skin is dry and its elasticity is diminished. The coat is rough and there is progressive wasting. Animals lose their energy, tire easily and show signs of broken wind.

The enlargement of the liver is only exceptionally sufficient to cause it to encroach upon the hypochondriac region. In the

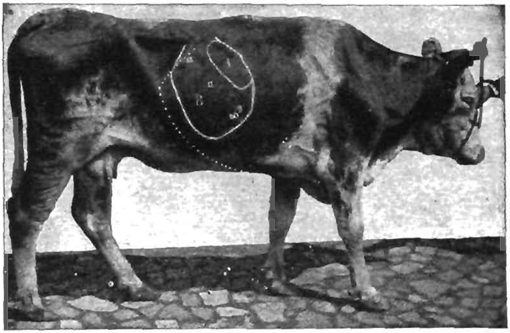


Fig. 81. Enlargement of the area of hepatic dulness in a cow due to chronic hepatitis. A. Normal area. B. Enlarged area. The dotted line marks the position of the costal arch and the numbers indicate the corresponding ribs. The liver weighed 14.6 kilos.

horse, carnivora and swine, this encroachment may be bilateral but in the ox it is only on the right side and then only as far as the anterior part of the hollow of the flank. Similarly it is only very exceptionally that the enlargement is sufficient to allow of palpation of the organ per rectum. In these cases it can be felt in the neighborhood of the last rib as a firm object lying against the abdominal wall and moving in concert with the respirations. Far more frequently in cattle the upper border of the enlarged liver can be felt by pressing inwards with the fingers behind the last rib. In carnivora and emaciated swine the liver can be felt through the abdominal wall under both costal arches, in calves and small ruminants under the right costal arch only. The hepatic area is very susceptible to pressure, animals experiencing difficulty and exercising great care in turning.

An increase in the area of hepatic dulness may be referred without any further delay in carnivora, ruminants and swine, to a somewhat pronounced enlargement of the liver. The same

holds good for the horse. In carnivora (Figs. 82 and 83) the enlargement extends downwards and backwards, usually reach-

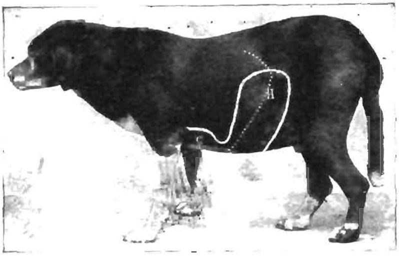


Fig. 82. Enlargement of the area of hepatic dulness in a dog due to chronic hepatitis. H. The enlarged area extending to the navel in the forward direction, as far as the cardiac area (C). The costal arch is indicated by the dotted line. The dog was of medium size, but the liver weighed 3 kilos.

ing the umbilical region. In ruminants (Fig. 81) it extends backwards and downwards to below the costal arch on the right side, or into the anterior portion of the hollow of the flank. In ruminants the normal area of dulness depends upon the quality of food in the stomachs and intestines. In the horse (Fig.

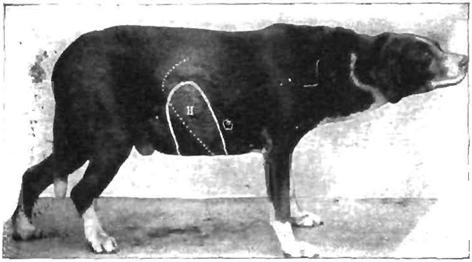


Fig. 83. Enlargement of the area of hepatic dulness in a dog due to chronic hepatitis. The right side shown in Fig. 82.

80) in cases where the enlargement of the liver is considerable, there is an area of dulness immediately behind the edge of the lung on the right side from the tenth to seventeenth intercostal spaces. This may extend downwards to below the costal arch. Under similar circumstances there is a smaller area on the left side below the edge of the lung from the eighth to the tenth intercostal spaces.

Marked reduction in the size of the liver is sometimes indicated by a decrease in the size of the area of hepatic dulness. In these cases in the smaller animals the liver can be felt from the costal arch only, the firm consistency and uneven surface can, however, be appreciated.

Fairly frequently, and especially in dogs, there is ascites which may persist without complications till death, or towards the end of the disease there may be edematous swelling of the abdominal wall and legs.

Enlargement of the spleen can be diagnosed in carnivora and emaciated pigs by palpation of the abdomen. In the horse it may sometimes be discovered by rectal examination.

In cattle, jaundice is frequently seen, and towards the end it is very severe. In other animals it usually sets in at a late stage and is not very pronounced even then. This is quite in accordance with the fact that the hyperplastic form of the disease is the commonest in cattle.

The specific gravity of the urine is higher, it is darker in color and contains less uric acid than normal; but in some cases albumin, sugar and urobilin are present. (In a liter of urine from a horse, Mouquet found 7.25 gm. of uric acid, 0.12 gm. of albumen and 15.4 gm. of sugar. Urobilin was also present.)

Towards the end of the disease, nervous symptoms make their appearance, the animals becoming dull and lethargic. In Schweinsberg disease and condition caused by plants of the order Senecio, these symptoms are sometimes very severe, and appear in the early stages. There is giddiness and unsteadiness of gait, the animals lean forward with their heads against the wall and attempt to go forwards, make gaping movements and seem dull and sleepy. The appearance is suggestive of staggers (Beichold, Imminger, Robertson).

In cases of secondary cirrhosis of the liver the symptoms of the primary disease are present.

Course. The disease lasts for several months (usually from three to six months in the case of Schweinsberg disease). The disease of the liver and the subsequent catarrh of the stomach and intestine cause a loss of condition, pronounced wasting and anemia. In the later stages there are often dropsical swellings. Finally in the last stages there may be hemorrhages in the mucous membranes, gums, skin, stomach or intestines. Exceptionally rupture of the liver or dilatation of the stomach (in the horse) may cause death unexpectedly, death usually occurring only after the animal has reached a state of utter exhaustion.

Diagnosis. In regions where cirrhosis of the liver occurs enzootically, the appearance of the peculiar digestive troubles permits the supposition that a chronic hepatitis exists; in its later course this can be recognized with certainty when persist-

ent icterus, enlargement or increased consistency of the liver exist, or isolated ascites, enlargement of the spleen, and nervous manifestations. A sporadic cirrhosis of the liver can be recognized only if the local changes, which have been described, are present, and in such a case mistakes are by no means impossible because other diseases of the liver may produce similar symptoms, more or less. If ascites exists, the exudate should be removed by puncture in order to facilitate a more exact examination of the liver after the abdominal walls are relaxed. The differentiation of hyperplastic and of atrophic cirrhosis is rarely possible with sufficient exactness. Aside from the change in form of the organ, and from the increased liver dullness, ascites with enlargement of the spleen suggests more the existence of atrophy, while very evident jaundice speaks for the presence of hyperplastic cirrhosis.

Large and firm nodes, associated with rapid and striking emaciation (cancer-cachexia) suggest primarily the presence of carcinoma of the liver, the diagnosis receiving support if a primary carcinoma is found in other organs (mammary gland, intestine, pancreas, lung), or if metastatic tumors are discovered in the lung.—In amyloid degeneration, the liver is enlarged uniformly, moderately firm but smooth, ascites is absent or at most exists with other hydremic symptoms; in a part of the cases diseases or injurious influences can be shown to have occurred, which are known to lead to amyloid degeneration.—Echinococcosis of the liver is characterized by comparatively large, elastic, tense or possibly fluctuating lumps which can be palpated in the enlarged liver; and by the fact that no severe digestive and nutritional disturbances need be present. In some cases exploratory puncture of the liver and the complement-fixation reaction may be of value.—Peritonitis or ascites originating independently of hepatic cirrhosis may give rise to a similar increase in circumference as a greatly enlarged cirrhotic liver; but they can be eliminated readily by establishing the horizontal borders of the dullness, and by the absence of enlargement of the liver.—Leukemic or pseudoleukemic enlargement of the organ is recognized by the other symptoms of leukemia or of pseudoleukemia (symmetrical enlargement of the lymph nodes, leukemic blood picture).

Prognosis. The disease is inevitably fatal, but there may be temporary improvements.

Treatment. In the first place the probable cause should be removed. The diet should receive careful attention, all sour grasses or irritating materials should be excluded, and a change of food should be given. Thus, a decided decrease in the occurrence of Schweinsberger disease was achieved in Bavaria by regulating the flow of the water courses and in this manner improving the feed conditions. In one community in Bavaria good results were obtained when the 6-year-old horses were

moved to pastures in noninfected districts for a longer or shorter period. Medical treatment promises little success. In stasis of the liver, a decided decrease in the volume can often be obtained with cardiotonic drugs after removal of the compression of the posterior vena cava, if such should be present, by thoracocentesis or by tapping the pericardial sac, etc. Imminger advises intratracheal injections of Lugol's solution in cases in the horse; others have had no good results from this treatment.

12. Neoplasms of the Liver. Neoplasmata hepatis.

Cancer of the Liver. Carcinoma hepatis.

Occurrence. Cancer of the liver is rare in all the domestic animals but among them it is most frequently seen in the dog.

Anatomical Changes. Primary cancer of the liver is usually adeno-carcinomatous in type. Secondary cancer of the liver generally follows primary growths in the stomach and intestines, in which cases the cancer cells appear columnar as a rule. The primary growths may also be situated in the peritoneum, mammary gland, pancreas or lungs. The lesions in the liver may vary in size from a millet seed to a fist or even larger. They are either white or pale red in color. In many cases they can be enucleated. The superficially placed growths may show a slight central depression (Krebsdelle). The cut surface shows a sulphur- or butter-yellow network. The consistency varies from that of marrow to that of fibrous tissue. Lesions in the livers of cattle are frequently soft, and contain a semi-fluid pulplike material, and in color they scarcely differ from the normal liver tissue (Kitt). The portal lymphatic glands are almost always affected. In cases of primary hepatic cancer, there is usually only a single large tumor, but metastatic lesions are almost always numerous. The weight of the organ is increased according to the number and size of the lesions. Chauvrat records a horse's liver weighing 21.6 kilos; van Tricht, the liver of a cow weighing 22 kilos, and Wilhelmi, one weighing 51 kilos.

Cases of cancer of the gall bladder have been observed (Gurlt, Bruckmüller, Kitt, Johne). The gall bladder is enlarged and nodular, the wall is thick and firm and the inner surface shows either pedunculated or sessile growths which at places are ulcerated.

Symptoms. Symptoms are first observed when the disease has made some progress. There is gradual wasting and anemia, the appetite is diminished, and in some cases there is vomiting. In a cow with carcinoma of the liver, Schlegel observed symptoms of pneumonia lasting two days and being asso-

ciated with high fever, which occurred in consequence of numerous recent carcinomatous metastases in the lungs. In the dog there is striking atrophy of the muscular system, the muscles of mastication being chiefly affected. This gives the animal a peculiar facial expression ("Krebsgesicht," "Facies cancéreux" [Trasbot]).

In ruminants and dogs an enlargement of the area of hepatic dulness can sometimes be demonstrated, or in small animals the enlarged liver may be felt below the costal arch. If small nodules can be discovered, the suspicion is at once aroused that the case may be one of cancer of the liver, especially if other lesions (such as carcinoma of the mammary gland) suggest that possibility. In some cases there is jaundice, and in others ascites and swelling of the spleen.

With reference to differential diagnosis tuberculous swellings must be considered, which often give rise in dogs to very similar clinical findings as carcinoma of the liver; the tuberculin test may be negative in these cases. It is also necessary to eliminate enlargement of the lymph nodes of the liver, swellings in the pyloric region of the stomach, in the pancreas, mesentery or in the omentum; also an enlargement in the circumference of the gall-bladder, and dilatation of the bile-ducts.

Course. Several months may elapse after the appearance of the first symptoms, but finally the gradually increasing cachexia leads to complete exhaustion.

Treatment. Spontaneous recovery never occurs and internal treatment is useless. No very satisfactory results are to be expected of surgical interference. (Parascandolo removed a carcinoma from the liver of a sheep with good results.)

Other Neoplasms in the Liver. From a clinical point of view these have only a subordinate interest as they generally remain unobserved during the life of the animal and can only be distinguished from carcinomata when the primary growth is situated in some part of the body or in some organ that is more accessible to investigation. Sarcoma of the liver most closely resembles carcinoma of that organ. Primary sarcoma of the liver is extremely rare, but metastases from growths in the peritoneum, intestine or mesentery are observed somewhat more frequently. In these cases also, there is a gradually progressive cachexia, enlargement and sensitiveness of the liver, and in some instances jaundice and ascites. Cadéac records a case in which the neoplasm involved the wall of the portal vein in an ox and caused the animal's death. In the liver of the horse melanomata and melanotic sarcomata are not absolutely rare and these may be present in large numbers.

Adenomata (Ad. simplex, fibrosum, Cholangiosum viride) are very rare and cause symptoms only when they become carcinomatous.

Angiomata are of fairly common occurrence in the ox, but rarely cause any symptoms. The lesions are generally numerous. (Hemangioma cavernosum or Telangiectasia capillaris.) Trasbot records a case in which a horse which had shown slight symptoms of colic

a year previously, died from hemorrhage following rupture of an angioma, and Ball saw a similar case in a cat.

Trasbot found two lipomata in the liver of a dog, one of which was as large as a child's head, and Ratz has seen lipomata in the livers of birds.

Villus-like outgrowths sometimes occur in the gall-bladder, especially in cattle. These sometimes are simple papillomata and sometimes have the structure of a villous cancer. In the latter case, secondary cancer nodules may be present in the liver.

Tuberculosis and Actinomycosis of the Liver. Tuberculosis of the liver is common in cattle, pigs and birds, more rare in the dog, and is exceptional in the other domestic animals. It is usually secondary to disease of the peritoneum, intestine or mesenteric glands. Occasionally it is embolic and it is also *primary in young animals that have been infected by way of* the placenta. The liver contains at first only quite small lesions and later caseous or caseo-purulent lesions of considerable size. The material contained in the lesions sometimes resembles mortar. In some cases the lesions have the appearance of lympho-sarcomata, and very often there is a marked increase in the weight of the organ. In the dog, the lesions sometimes have a central depression, and thus to some extent resemble cancer growths. In sucking animals, it is more or less likely that the disease will only be recognized if, in addition to tuberculous lesions in other organs, enlargement of the liver and a nodular condition of that organ can be discovered.

The lesions of actinomycosis are similar except that they consist of a fibrous capsule enclosing a yellowish, soft, gelatinous tissue which is becoming purulent at places, and in which the actinomyces can be found by microscopic examination.

13. Animal Parasites of the Liver.

(a) Echinococci.

Occurrence. In cattle and pigs echinococci are of very frequent *occurrence but rare in solipeds and carnivora.* The parasite is also found in man and in turkeys.

The distribution of echinococcosis is intimately related to the number of dogs and the frequency with which they are hosts of the tænia echinococcus. In Ireland and, according to Vidal, in Tunis also there is scarcely an adult ox or sheep that does not harbor echinococci. The disease is also widespread in Australia and India (70% of all bovines). In Europe, Mecklenburg appears to be affected most seriously. Madelung and Sahlmann found 25 to 50% of cattle, 75% of sheep and 5 to 8% of pigs infested. Metelmann found echinococci in 25% of cattle, 15% of sheep and 5% of pigs.

According to the statistics of 52 German slaughter houses drawn up by Peiper 11% of cattle, 10% of sheep and 6.5% of pigs were affected. In 1896-97 at the Berlin abattoirs the livers of 1,156 cattle, 1,939 sheep and 5,398 pigs were seized out of a total number of 146,612 cattle, 395,769 sheep and 694,170 pigs (Ostertag). According to Längrich the percentages of animals found to be infested at the abattoirs at Rostock were 36% cattle, 26.5% sheep and 5% pigs. In goats and horses the number was 1%. At Stettin, Olt found 7.1% of cattle, 26.8%

of sheep and 7.3% of pigs affected. Schmidt calculates the annual loss in cattle to amount to $37,500. At Budapest during the period 1899-1903 out of 511,031 cattle killed (including 20,000 buffaloes) there were 7,622 cases or 1.4%; 3,755 cases were found among 90,883 sheep (4.1%), and 5,105 out of 474,401 pigs or 1.1%. At Prague the percentage of affected cattle amounted to 23.2% and of sheep 5.5% (Prettner). In Russia the incidence of the parasite varies from 0.1 to 80% in cattle, 0.01 to 60% in sheep, from 0.01 to 70% in pigs and from 0.005 to 40% in horses.

Lichtenfeld's investigations regarding the distribution of the parasite in the various organs are very interesting. These showed that the lungs were affected in cattle in 69.3% of cases, the liver in 27%, the spleen 2.2%, heart 0.75% and kidneys in 0.75%. In the sheep the figures were: lungs 52.2%, liver 44.9%, spleen 2.9%. In the pig the figures were: lungs 18.8%, liver 73.1%, spleen 2.95%, and heart and kidneys in 2.25%. In 1.3% of cases there were parasites in the subperitoneal tissue. In the horse the lungs were involved in 5.5%, and the liver in 94.5% of cases. It was also shown that in pigs under two years old the proportion between the number of cases in which the lungs were affected and those in which the liver was affected is 12.8:82. In adult animals the proportion was 39.3:46.4.

Echinococcus disease is comparatively frequent in man. In the central parts of Europe 1 case is seen in 130 postmortems. Echinococcus multilocularis is practically confined to places where cattle are principally affected (Tyrol, Inntal and the Memmingen district in Bavaria), whereas E. polymorphus is seen in those places where sheep are the principal hosts (Iceland, Australia, Mecklenburg, Pomerania, Dalmatia, Argentine).

Etiology. Echinococci develop from the embryos of the tænia echinococcus (Siebold) which inhabits the intestine of the dog. These embryos are liberated from ingested eggs in the stomach and pass out of the intestine into the portal circulation. The vehicles of infection are food or water contaminated with the feces of dogs or with proglottides or tapeworm eggs contained in these.

Evolution of Echinococci. The oncosphere of the echinococcus-taenia, which has been carried in the circulation, changes, in the organs that are suitable for its further development, into a very small cyst (hydatis) which gradually becomes larger; its wall consists of two layers, the outer, thicker chitinous layer having a characteristic lamellate structure, while the inner germinal or parenchyma layer, is much thinner, of cellular structure and of correspondingly granular appearance. Such a cyst is surrounded by an organic skin formed by the organ of the host (host-capsule) which consists in fertile echinococci, almost entirely of fibrous connective tissue, but in sterile echinococci of young connective tissue cells within and outside of these, of round cells; further of fibrous connective tissue; it contains varying numbers of eosinophile leucocytes and, in sterile cysts, giant cells (Gasse). The fluid of the cyst is clear as water or slightly yellowish; its specific gravity is 1007 to 1015 and it contains a large amount of succinic acid. Within the cyst the germinal layer forms small brood-capsules, by budding, which consist of a fine membrane, imitating the structure of the germinal layer, and enclosing 3-10 scolices; they can separate from the cyst and then swim free in the content of the echinococcus. Even the single scolices may become free through opening of the brood-capsules. The budding of the germinal layer also leads to the development of minute daughter-cysts similar in structure to the original cyst which has now become the mother-cyst, consisting of lamellated cuticula and germinal layer. The daughter-cysts grow toward the lumen of the mother-cyst, finally falling into its liquid contents and enlarging in it, where they

also form brood-capsules. It even happens that "granddaughter-cysts" develop. The echinococcus which forms these endogenous daughter-cysts, is called Echinococcus endogenus (s. hydatidosus). In the E. exogenus s. granulosus the daughter-cysts grow toward the periphery of the mother-cyst (exogenous daughter- and granddaughter-cysts), they cause a bulging of its surface and finally part from it, although they remain in touch with the mother-cyst, the whole giving rise to a grape-like formation.

In contrast to this process in the E. fertilis, no brood-capsules form in the E. sterilis (Acephalocysta), although the hydatid may attain a considerable size and form daughter- and granddaughter-cysts. Litchenfeld found a larger number of fertile cysts in pigs under two years old than in older animals, and he also found that the proportion existing between fertile and sterile cysts was in cattle 24:76; in pigs 80:20; in sheep 92.5:7.5 and in horses 38.9:61.1.

According to the investigations of Bobroff, v. Alexinsky, Dévé and Ponomaroff, a shorter evolution of the echinococcus may take place, if a fertile cyst bursts and liberates scolices, brood-capsules or fertile daughter-cysts which are then carried with the lymph stream into other portions of the affected organs, or with the blood-stream into other organs where they may form emboli. The fertile echinococcus cysts then develop directly from these scolices which have been transported in this manner (Echinococcus secundarius).

The development of echinococci is very slow. According to Leuckart small foci measuring 0.25 to 0.35 mm. in diameter and enclosed in thin fibrous capsules are found one month after infection. After two months they measure about 2.5 mm. and are already converted into small cysts. Towards the end of the fifth month the cysts are about the size of hazelnuts, the two layers of the wall are well differentiated and the development of the scolices and daughter-cysts has begun.

The Echinococcus alveolaris s. multilocularis always grows through the exogenous formation of cysts which extend preferably into the preformed hollow spaces of the biliary passages, of the blood- and lymph vessels, and grow at their expense to not more than pea-size. According to Ostertag, Mueller, Mangold and Posselt this form of the echinococcus corresponds to a particular species of taenia, the Taenia echinococcus alevolaris, but this is doubted by Klemm and Linstow. Neveu-Lemaire claims that the E. multilocularis should be separated from the true E. alveolaris as he considers the former merely as a peculiar form of the E. polymorphus with exogenous cyst-development.

The death of the echinococci with subsequent inspissation, caseation and calcification, or with suppuration of the contents may occur in any stage of the evolution; according to Mehlhose it is due to a purulent, fibrinous or productive inflammation of the organic covering, under the influence of bacteria which have been transported, with the oncospheres, from the intestines of the intermediary host.

Pathogenesis. The immigration of the brood takes place through the walls of stomach and small intestine in such a manner that the covering of the ingested tænia eggs is dissolved, the oncospheres are liberated and then pierce the mucous membrane with the aid of their hooks; they enter the branches of the portal vein and are carried into the liver with the portal circulation, being arrested partly in the capillaries. The smaller

oncospheres pass through the capillaries of the portal systen get into the right heart and into the pulmonary circulation an are arrested there in greater part. Some of the oncosphere can pass even the pulmonary capillaries and be carried int different organs with the blood of the greater circulation. Afte having localized in an organ, the oncospheres develop int echinococcus cysts in the manner described above.

In contrast to their frequency, the echinococci only rarel cause disturbances in health, even if their number is conside able. Only after an excessive invasion, and if only isolate small islands of normal liver parenchyma remain, acholia occur associated with digestive troubles, in which the greatly enlarge liver impedes the motions of the diaphragm, and possibly inter feres with the functions of the abdominal organs, especially th stomach and intestine. Echinococci near the entrance of the por tal artery may produce compression of this artery (Lucas), i they are near the dorsal border of the liver, they can caus compression of the esophagus (Weber).

On being absorbed into the circulation the specific albumin of the hydatid flui gives rise to the formation of specific antibodies in the body of the host, which ca be demonstrated serologically, and may also perhaps produce anaphylaxis; if the greater amounts of echinococcus fluid are absorbed accidentally, as after burstin of a cyst during operation or during exploratory incision, an anaphylactic shock ma be produced in the shape of hydatid-poisoning (Pfeiler). This may in part accour for the many contradictory findings concerning the toxicity of the echinococcus flui in which Mourzon and Schlagdenhaufen, for instance, claim to have found a toxa bumin, while it was shown to be non-toxic in Joest's animal experiments. The toxi action which the hydatid fluid may show occasionally, may also be due to a laten infection with different bacteria (Gherardini), staphylococci, streptococci, bacteriur coli, micrococcus tetragenus, sarcina lutea, proteus bacilli, bipolar and diphtheria-lik rods having been found in it (Griglio, Mehlhose).

Anatomical Changes. The liver is enlarged in proportior to the size and number of the cysts present in it, and in th ox may weigh as much as 158 pounds (Ringk). In the pig, in fested livers are sometimes of enormous size (110 pounds, Gé rard). The surface shows rounded prominences of various sizes and there may be a considerable distortion of the organ. If a cyst be cut into, there is a prompt escape of a clear, yellowisl serous liquid and a rounded cavity is exposed. The interior o the connective tissue capsule is lined by an elastic membrane which is easily detached and torn, and tends to roll up. This is the true cyst wall and bears on its surface small whitish promi- nences, the brood capsules, and it may also contain severa daughter cysts and granddaughter cysts. The liver tissue ir the immediate neighborhood is very pale in color, and if nu- merous cysts are present, develops fine but tough strands o connective tissue. In dead cysts there is either a soft and pulpy or a caseous mass in which shreds of the cyst wall can be found, and sometimes chitin-hooks. This result and the ab- sence of young tubercles in the surrounding tissue and in the lymph nodes of the liver differentiate these foci from caseated tuberculous foci. In case of suppuration of the echinococcus,

portions of the laminated cyst wall, and sometimes hooks are found in the content of the abscess.

The Echinococcus alveolaris forms more tumorlike masses than the E. polymorphus, the surface of the former being beset with cysts. On cross section, these bear considerable resemblance to alveolar or colloid cancers and have the structure already described. Formerly such lesions were considered as cancers. Their true nature was first discovered by Virchow in 1855.

Similar cysts are found in the lungs and more rarely in other organs.

Symptoms. In **cattle** digestive disturbances, lasting for weeks or months, have been observed in some cases: capricious appetite, inactivity of rumination, constipation, frequent tympanitis, etc. Exceptionally jaundice and wasting and the general symptoms of ill-health have been seen. The area of dulness may be greatly increased and may extend backwards to the last rib and in the downward direction to the lower third of the abdomen (see fig. 81, page 457). Palpation of this region may cause symptoms of pain. The enlargement of the liver may be so great that the right half of the abdomen and right flank are rendered prominent. By manipulation of the right side of the abdominal wall, and by rectal examination, one can, in cases where the enlargement of the liver is excessive, feel the thickened and rounded edge of the liver and the elastic nature of the prominences can be appreciated (Labarrère, Ländler). Respiration is usually shallow and accelerated. Weinberg and Vieillard found that echinococcosis can generally be diagnosed by the fixation of complement method.

The symptoms in the **sheep** are similar to those described. The disease can only be distinguished from the similar condition caused by flukes if the unevenness of the surface of the liver can be felt through the abdominal wall.

In **pigs** Lucas also saw pronounced ascites in cases of heavy infestation. The animals were quite unable to get onto their hind feet and dragged them behind them. In spite of a good appetite, there was marked wasting. In a case recorded by Schmidt, there was evidence of jaundice, while in one published by Friedrich there was enormous distension of the abdomen without any ascites.

Diagnosis. The diagnosis is based upon the palpation of elastic, tense or fluctuating, smooth and spherical swellings in the greatly enlarged liver, the absence of fever and the fact that the nutritional disturbances are slight in proportion to the liver enlargement. Under certain circumstances it may be advisable to attempt an exploratory puncture of the palpable swellings, in order to demonstrate shreds of membrane, scolices or hooks in the fluid. In man, symptoms of poisoning have been observed frequently after exploratory puncture, especially gen-

eral debility, dyspnea, collapse, eclamptiform convulsions, increase or reduction of the body temperature, papular eruptions and arthralgia, which probably occurred because of a partial absorption of the echinococcus fluid.

In the more frequent cases in which the liver is enlarged only slightly, echinococcosis may probably be determined clinically only by means of the complement-fixation test. Weinberg & Vieillard, Putzu and others found this method to be very reliable although not absolutely certain.

Sparapani detected such antibodies in calves from cows suffering with echinococci, which, however, soon disappeared after birth and hence could only have been derived from the mother through the placenta. They used as antigen the clear sterile echinococcus fluid which had been withdrawn from slaughtered animals and which is best extracted with ether, the fluid itself being only slightly active especially in animals (Falcoianu). On the other hand, no decisive diagnostic importance can be attributed to the precipitin test (Pfeiler). Ascoli's meiostagmin reaction also gave positive results (Silva).

Treatment and Prophylaxis. No satisfactory method of treating echinococcosis is known. Since the disease is set up by the ingestion of the eggs of the Tænia echinococcus, animals should be prevented from eating food or drinking water that are soiled with the feces of dogs. As this is generally impracticable care must be taken that all organs of animals slaughtered that contain echinococci are destroyed and not, as so often happens, given to the dogs.

(b) Distomatosis. Fluke Disease.

(Liver rot.)

The disease is caused by the Distomum hepaticum and D. lanceolatum. It is an acute or chronic inflammation of the liver and bile ducts, more commonly the latter. In the chronic form it leads to serious loss of condition.

Historical. The earliest records of the disease are those of Schaper von Gabucinus in 1547, and of Gemma a few years later. The term, "Leheregel" (fluke) was first used in 1676 by Frommann. Valuable clinical and pathological studies of the disease were made by Schäffer (1764), Goeze (1782), Chabert (1879), and Bilhuber (1791). The life-history of the parasite was elucidated by Mehlis (1831), v. Nordmann (1832), Eschericht (1841), Steenstrup (1842), and especially by Leuckart (1876), who determined the metagenesis of the fluke. Among veterinary surgeons the names of Gerlach (1854), Delafond (1854), Davaine (1860), Friedberger (1878), Zündel (1880), Thomas (1881), and Zürn (1882), are closely connected with the study of distomatosis. Interesting investigations were made by Schaper (1889), and Lutz (1892), regarding natural infection, and also by Schaper in connection

with the pathogenesis, symptomatology and pathological anatomy of the disease.

Occurrence. The Distomum hepaticum is found in marshy places everywhere, whereas the distribution of the D. lanceolatum appears to be more restricted, the parasite being far more common in southern Europe than in northern. The geographical distribution of the D. hepaticum exactly coincides with that of Limnæus minutus, which is practically the exclusive intermediate host of the parasite (Leuckart). After wet summers the disease is very widespread and causes very heavy losses. The severe form of the disease rarely occurs among adult cattle, although they may harbor the parasite in their livers. It is principally among calves that serious losses occur. Extensive outbreaks of the disease sometimes occur among goats (Römer). Swine, buffaloes (Hungary and East Indies), camels, wild ruminants, hares and exceptionally horses, asses, dogs, cats and rabbits are infested. Liver rot occurs very rarely in man. In isolated cases, more or less developed or even mature specimens of D. hepaticum have been found in newborn calves, one to three days old, in which the biliary passages were altered only slightly (de Jong, Buechli, Waldmann), also in sucking lambs and even in a fetus (Billhuber, Bloch, Frommann, Schlegel).

In 1873 a third of the sheep, which were valued at more than $200,000, died in Alsace-Lorraine. In England the annual loss is computed at a million sheep. Great losses were caused by fluke in Hungary and especially Upper Hungary in 1889. On two farms practically the entire flocks were wiped out. The disease was also very prevalent during the years 1893-1897. According to Popow in certain districts of Poland in 1891 from 50 to 90% of the sheep died. The liver fluke is not known in Iceland (Krabbe).

In 1876 the disease caused losses up to 40% of the cattle in Slavonia. The disease is frequent in Asia, in America and in Australia.

In 1883-84 36% of all the cattle, 7% of the calves and 1½% of the pigs killed at the Berlin abattoirs were found to be affected. At Budapest during the period 1889-1903 the percentages of affected animals were: 4.9% of cattle, 39.5% of sheep and 25% of goats. In 1902-03, 474,401 pigs were slaughtered and of these 1.2% were seriously infested. At Okayama (Japan) Saito found the Distomum hepaticum in 16% of cattle but the lesions were confined to the bile ducts.

Etiology. The disease is caused by two varieties of distomes belonging to the Trematodes family of the Platyhelminthes. These two parasites are called Distomum hepaticum (Fasciola hepatica) and D. lanceolatum (F. lanceolata, Dicrocœlium lanceolatum). In some regions other distomas are responsible for the affection. Both parasites are leaf-shaped, elongated and unprovided with appendages. The body is broader towards the middle part and there are two suckers at the anterior end; they are hermaphrodites.

Distomum hepaticum measures from 20 to 30 mm. in length by 3 to 13 mm. in width, and is covered with minute spines that point backwards. At the anterior extremity there is a conical process which carries at its apex the oral sucker. On the under surface, about 3 mm. behind this, there is a second and larger sucker.

The eggs (fig. 87) are brown or greenish-yellow, oval, and provided at one pole with a small cap. This cap may be rendered visible either by exerting pressure on the eggs or by the addition of potassium hydrate solution. The eggs measure 130 to 145 μ in length and 70 to 90 μ in width.

The Distomum lanceolatum is only 4 to 9 mm. in length and 2.5 mm. broad. It is slender and lancet-shaped. The eggs which are brownish in color are also provided with a polar cap (fig. 86) and measure 37 to 40 μ in length.

Further, Fasciola magna has been found in North America in various cervides, also in sheep, goats, horses and cattle; it differs from the common liver-fluke in that the latter is larger and its eggs have larger dimensions; similarly the more slender F. gigantea which is found in Africa in buffaloes, cattle, sheep and goats, also in giraffes and zebras. F. aegyptica was found in Egypt in cattle, buffaloes, sheep and goats; F. angusta in cattle in Senegal (Neveu-Lemaire).

Fig. 85.

Fig. 86.

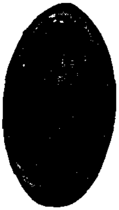


Fig. 85. Egg of Distomum lanceolatum.
Fig. 86. Egg of Distomum hepaticum.

Fig. 87. Distomum hepaticum (natürl. Gr.).

Fig. 88. Distomum hepaticum (makroskopischer Bau, doppelte Gr.).

Fig. 89. Dist. lanceolatum (natürl. Gr.).

Life-cycle of the Distomum Hepaticum. According to Weinland, Leuckart, and Thomas, the life-cycle of the parasite is as follows: The eggs of the mature flukes are passed out with the feces of the host into the outer world. Provided segmentation has taken place, develops during the next 3 to 6 weeks, in the presence of sufficient warmth (at least 10-12° C., 50-54° F.) and moisture, a closely ciliated elongated embryo (miracidium) with X-shaped eye spots. In water, or in material containing much water, and in the presence of light, the polar cap of the eggshell is forced open, the embryo escapes and begins to move actively about, according to Leuckart for three hours, according to Thomas for one day, in alkaline fluid even for three days. If in the meantime it finds a suitable host, especially the fresh-water snail, Limnæa truncatula s. minuta (Limnius minutus s. truncatulus), or in the absence of this, other species of Limnæa (in America, L. humilis, L. oahuensis, and L. viator; in the Sandwich Islands, L. rubella; in Australia, L. tenuistriata; in Africa, L. natalensis or Physopsis Africana), it penetrates into their respiratory cavity, and from there (or seldom from the surface of the body) into the body tissues, sheds its ciliated skin, and transforms into a tube-like sporocyst with an intestinal tract. In the following 2 to 4 weeks the sporocyst divides into 5 to 8 rediæ (elongated, cylindrical, with the digestive tract closed at one end), and from these daughter rediæ are formed while the snail hibernates

during the winter. In from 4 to 6 weeks more there are formed in each redia from 15 to 20 oval, tadpole-like cercariæ with two suckers, long rudder tail and branching intestinal tract, which now emerge through the oral cavity of the rediæ and later also leave the body of the snail to swim about in water or crawl about on very moist objects. Soon they attach themselves, after casting off the rudder tail and excreting a sticky mass that dries in a few minutes, to stems of grass, leaves of shrubs standing in water, and even to the shells of snails, and convert themselves into whitish cysts resembling grains of sand and measuring 2-3 mm. According to the investigations of Ssinitzin, the encapsulation takes places 1½ cm. below the water level in an average of 66% of the cercariæ; in 24% they are located deeper and in 10% above the surface of the water, while in 6% they encapsulate in a free state directly on the surface of the water. In the latter case an outer membrane is formed which is dilated at the top and filled with air, thus serving to keep the encapsulated cercariæ afloat. Sometimes encapsulated cercariæ are even found in the body of the Limnæa. Only after encapsulation has taken place will they become infective. The membrane of the cysts is dissolved in the stomach of a suitable host, whereupon the cercariæ, measuring about 1 mm., migrate to the bile ducts of the host, and there, according to the observations of Railliet, Moussu and Henry, and those of the author, become sexually mature in about three months (Pozajic found distoma 1½ cm. long in an 8-weeks-old colt). The observations to the contrary by Gerlach, Friedberger and Leuckart, according to which the period of development is three weeks, and those of Thomas, reporting a period of 5 to 6 weeks, and based upon the presence of mature distoma together with entirely undeveloped specimens 3 and 5 to 6 weeks after the animals were removed from pasture, can be explained by the fact that the invasion took place at different periods during the entire time that the animals were at pasture, the mature specimens therefore originating from an earlier infestation. According to Buechli and de Jong, the distoma can mature in a few days, since mature distoma were found in calves 3 days old and are said to have been derived from an extrauterine infestation. The occurrence of distoma in feti also permits the conclusion that an intrauterine infestation may take place.

The flukes remain in the liver for a period of from 3 to 5 years. Railliet, Moussu and Henry found them after 3 years, and Thomas after five and one-quarter years in animals that had no opportunity to become reinfested during this period. The majority of the flukes, however, leave the liver in from 9 months to 1 year, and the emigration takes place during any season of the year, corresponding to the possibility of infestation, which lasts all the year round, and to the longevity of the different flukes. This explains the observations of Friedberger and Pech, according to which emigrating flukes were found in December—that is, from 3 to 4 weeks after the termination of the period of pasturage. On the other hand, some flukes die and disintegrate in the bile ducts after oviposition; others migrate into the intestinal tract, where they die and are digested; while some reach the outside world in a well-preserved state.

The following observations point to a relatively slow growth of the immigrated cercariæ: Friedberger found in two lambs, on the forty-eighth and fifty-first days after their removal from pasture, young flukes 4-5-8 mm. long, besides mature flukes. Thomas found in a lamb harboring more than 200 flukes, on the seventh day, two that were only 1.1 to

1.5 mm. long, many hardly more than 2 mm. long, and a few that were 8 to 8.5 mm. long. The latter had probably immigrated at an earlier period. In another lamb he found on the nineteenth day flukes that were only 2 mm. long, as well as larger ones.

Limnæa truncatula or the mud snail (Fig. 89) has a spiral and often brown shell ½ cm. long. It is found especially on marshy ground, in ditches, in pools in which grass but no reeds grow, and in slowly running brooks. In fall it seeks water, mostly ditches with loamy soil, where it hibernates. The closely related Limnæa peregra is somewhat larger and the last spiral of the shell is somewhat more bulging. Leuckart succeeded in infesting young specimens of this species with fluke embryos which developed into sporozoites and rediæ, but since the sporozoites and rediæ later always perished, Limnæa peregra can hardly be considered as an intermediate host.

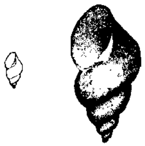


Fig. 89. Limnæa truncatula; left in natural size.

The life-cycle of the D. lanceolatum is in all probability similar but it is not yet known definitely. According to Piana, the pear-shaped larvæ penetrate the snail known as Helix carthusiana, and there develop into cercariæ. Leuckart believes that the escape of the embryos into the outer world is not spontaneous, but occurs in the intestine of a special intermediate host. According to Neveu-Lemaire, the further development into rediae and cercariae occurs in Planorbis marginatus or complanatus.

Resistance of the Parasite. The eggs remain capable of germinating from one year to another but do not resist drying or putrefaction. The larvæ, according to Friedberger, are not killed by night frosts, and the encapsuled cercariæ retain their power of development for weeks or even months (Leuckart). Salt solutions are very poisonous to the larvæ and cercariæ (Ercolani and Perroncito). A 2% solution is fatal in 5 minutes and a 1% solution in 20 to 35 minutes. Railliet, Moussu and Henry found that slaked lime was even more poisonous, killing the miracidiæ in a solution of only 0.5 per cent.

Infection. As a rule infection results from the ingestion of green food carrying encysted cercariæ. This is especially likely to happen on moist and marshy land, and on land that is frequently flooded. Contaminated water must also be responsible in many cases. According to Lutz, the latter method is the common one in the Sandwich Islands. The cercariæ becoming detached from water-plants in shallow streams collect at places where the water is shallow, and are then taken up by the cattle when drinking. As some cercariæ tend to become encysted in the bodies of grass snails, infection may take place through the

ingestion at pasture of plants bearing them. Spinola succeeded in infecting healthy sheep by feeding them with grass snails. Railliet, Moussu & Henry had the same experience with a lamb.

Housed animals may be infected, infection generally taking place through cercaria-infected green food, and sometimes through food from marshy land which has not been stored very long.

The infectivity of the pasture is maintained by eggs of live flukes that have retained their hatchability and by the rediæ and cercariæ that have passed the winter in the body of the snail, but especially by the yearly repeated reinfestation of the pasture by apparently healthy or recovered carriers, since, corresponding to the relatively great longevity of the flukes, such animals may pass fluke eggs with their feces for several years. Sometimes undomesticated ruminants and rabbits that harbor flukes may play a part in keeping up the infectivity of the pasture. Meadows may also become infested through the manure from carriers kept in the barn. When it is considered that the intermediate generations propagate asexually in the body of the snail, and that under the most favorable conditions from 150 to 300 or more cercariæ may develop from each egg, and 10 million from each fluke, it is not surprising that wet pastures may, under certain conditions, become heavily infested by even a few carriers.

The degree of infestation of the animals at pasture depends to a great extent upon the number of encapsulated cercariæ on the grass and in the water and upon the amount of moisture in the ground. Under normal weather conditions, only wet and swampy pastures, ditches with grass growing in them, and the banks of slowly flowing brooks are dangerous. However, in wet years, or in regions with a moist climate, the places that are already normally dangerous places not only become more dangerous, but regions that are normally free from infestation may become infested with eggs and cercariæ as well as snails, these being carried there by rains or by overflows. Under such conditions the snails will seek such places of their own accord. During wet years the pastures and meadows may also become more dangerously infested by reason of the continued change of the level of the water, whereby the stalks of grass will become infested throughout their entire length with cercariæ which become encapsulated at different times. This explains the experience that the liver-fluke disease may occur during wet years and become epizootic or even panzootic over wide areas.

The degree of infestation of the animals is also influenced by the length of time that they graze upon dangerous pastures. Sometimes a grazing period of only a few hours or even less than an hour may result in the infestation of a large number of animals. As a rule, however, a massive invasion takes place only after repeated grazing over infested pastures. In animals

fed in the barn only a medium degree of infestation probably occurs, except when such an infestation takes place through green feed, since up to the time that the feeding of hay begins in winter the majority of the cercariæ infesting the hay have perished. Only a few cercariæ are found in the fetus in cases of intrauterine infestation.

According to the observations of Friedberger and Schaper, infestation may take place at any time of the year, and accordingly may be repeated several times in the same animals; thus particularly in spring at pasture, or in winter in the barn in case relatively new hay or roughage that has ben preserved in a moist condition is fed, or when the animals are allowed to graze in the open during favorable weather. Massive invasion causing severe symptoms, however, occurs only during certain seasons of the year; in dry or normal years especially in the fall, in wet years also in summer. This is explained by the biological characteristics of the flukes. Eggs that have successfully passed the winter, or that have been disseminated in early spring over the pastures by carriers, corresponding to the 2 to 4 months' period of evolution of the intermediate generations, develop into infective cercariæ only toward the end of July or August. During normal years, when there is less rainfall during this period, the evolution is frequently at a standstill, as is also the cycle of evolution from eggs deposited at a later period. The slumbering evolution can proceed uninterruptedly only after sufficient precipitation in the fall and may then give rise to massive invasions. On the other hand, however, in wet years great numbers of encapsulated cercariæ may be found on forage plants during the second half of June.

Susceptibility. Sheep and wild ruminants are the most susceptible, the ox is a little less so and the goat a little less than the ox. Swine, solipeds, and carnivora on the other hand rarely become diseased in spite of being invaded by the parasite. Young adult animals are more susceptible or their powers of resistance are less.

The wide distribution of the disease among sheep convinced Railliet that the cercariæ are to be found principally on the lower blades of grass which the sheep eat, whereas cattle get only the upper ones. According to Humble and Lush sheep with short lower jaws (parrot-mouthed) escape infection because they can only bite the tips of the leaves.

Pathogenesis. It has been experimentally proved that the cercariæ of Distomum hepaticum may reach the liver by penetrating Glisson's capsule after perforating the wall of the intestinal tract and migrating around in the abdominal cavity for some time. The mode of immigrating into the liver already assumed by Gerlach, May and Spinola has been further substantiated by the experiments of Ssinitzin. On the other hand, according to the experiments of Railliet, Moussu and Henry

with Limnaeæ harboring cercariæ, it can not be denied that they may be passively carried to the liver by the blood stream of the portal vein in case the cercariæ, while penetrating the intestinal wall, should happen to get into one of its branches. In cases where only a light invasion has taken place, this may be the only mode of immigration, because under such circumstances points of penetration of the liver capsules are frequently absent in fresh cases. It is also possible that a few cercariæ migrate up the gall ducts if they happen to become lodged in the opening of the bile duct at the time that the intestinal contents pass this opening. Many authors assume that this is the only possible way they can reach the liver.

Following artificial infestation of rabbits, Ssinitzin observed that the ingested encapsulated cercariæ are carried into the intestinal tract and that the capsule is dissolved in from 2 to 3 hours, liberating the cercariæ. Later they penetrate the intestinal wall and reach the abdominal cavity, crawl about on the different organs for from 4 to 14 days, feed upon the red blood corpuscles which they derive from the superficial capillaries of the organs, in the same way that they later suck blood from the bile ducts. Owing to the loose structure and the large amount of blood contained in the liver, this organ is best suited for the nutrition of the young flukes. They therefore gradually collect upon the surface of the liver after the fourth day, attach themselves to it, bore into it, and gradually penetrate deeper along the bile ducts, disappearing from the abdominal cavity about 14 days after ingestion. The well-developed oral sucker and the hard, sharp spines covering the whole body at this stage of development are of great aid in penetrating the intestinal wall and the capsule of the liver as well as the liver tissue. During the first four days young flukes were never found in the liver, but only in the abdominal cavity, as in the cases of Sohns in guinea pigs and rabbits following natural infestation. This explains the negative results obtained by Leuckart in which the liver of a rabbit was examined soon after the ingestion of cercariæ.

While the young flukes migrate around in the liver searching for the bile ducts, the liver tissue and the bile ducts coursing in the same are destroyed, thus producing extensive tunnels which often reach the subserosa and may even perforate the liver capsule. Such perforations, however, are also produced when the cercariæ originally perforate the capsule from without. Through the mechanical irritation and the destruction of liver tissue, the gall ducts and liver tissue become inflamed and hemorrhages occur. Even abscesses may be formed, from which Schaper isolated streptococci and staphylococci. A few of the young flukes may also penetrate the hepatic veins, be carried into the right heart and the lungs and from there into other

organs. Thus in pregnant animals they may also reach the liver of the fetus by way of the placental circulation.

The young flukes which continue to wander about in the liver tissue probably perish if they do not succeed in finding and penetrating the bile duct necessary for their further development. As a result of the mechanical irritation by the spines on the surface of the body, the repeated attachment with the suckers, and the movement of the young flukes, a chronic inflammation with connective-tissue proliferation is set up in the walls of the bile ducts, which inflammation later often spreads to the interlobular connective tissue, thus producing cirrhosis of the liver. The destruction of the liver tissue soon after the penetration of the young flukes also is a factor in the production of cirrhosis. An important rôle is also played by the toxic products of metabolism and the excretions of the flukes, which, according to Guerrini, are excreted by a subcuticular excretory apparatus. The irritation by these substances is not only directed against the walls of the bile ducts, but also, after having been absorbed by the lymph stream, against the liver tissue. Bacteria, especially colon bacilli, probably also play an important rôle, finding suitable conditions for multiplication in the diseased bile duct after having been carried there by the blood stream, or after migrating into the gall ducts.

According to the investigations of Jaeger and Joest, the irritation due to the absorption of the toxins produces a serocellular infiltration at the point where the angles of the lobuli meet. This is followed by connective-tissue proliferation which gradually extends away from the bile ducts into the interlobular tissue, while the bile ducts actively proliferate by budding (also observed by Schaper); in short, a cholangioitic cirrhosis of the liver develops. The extensive cirrhosis of the liver is of less significance in the production of the ever-present disturbances of nutrition and blood formation as compared with the continued absorption of toxins from flukes and the bacteria localized in the bile ducts, as well as the blood-sucking activity of the liver flukes (proved by an observation of Railliet). This is substantiated by the observation of improvement in health and recovery following the destruction of the liver flukes in spite of the presence of cirrhosis of the liver. Distomatosis may also increase the susceptibility for specific and common infectious diseases.

Nothing definite is known concerning the mode and method of immigration of Distomum lanceolatum. The cercariæ of his group probably reach the liver in the same manner as those of the large fluke, and, in accordance with its smaller size and slender body, penetrate to the smallest bile ducts. According to he observations of Friedberger, Neumann, and the author, hese flukes have, as compared with Distomum hepaticum, a

less harmful effect, probably because they do not seem to be blood suckers and also because they cause a less severe mechanical irritation of the bile ducts (smaller size, spineless body surface). For this reason the inflammation is usually confined to the bile ducts, and even if a cirrhosis of the liver has developed in cases of massive invasion, no influence upon the organism as a whole is observed.

Anatomical Changes. If the invasion is extensive the liver shows evidence of an acute inflammation (Hepatitis acuta traumatica distomatosa). The liver is enlarged, hyperemic, its serous membrane is studded with small hemorrhages, and may be covered with a thin fibrinous deposit. The capsule shows here and there small, round, sharply-defined openings through which a dirty red liquid exudes on pressure, and sometimes the anterior extremity of a fluke may appear. These openings lead into irregular cavities in the liver tissue which contain blood, liver débris and numbers of young flukes embedded in a pulpy mass. If the liver be cut into, similar cavities are found in other parts. The large bile ducts are dilated and contain large quantities of muco-sanguinous bile and flukes in varying numbers. Exceptionally parasites escape into the peritoneum and set up a severe peritonitis, or there may be severe hemorrhage from the punctures caused by their escape.

In the livers of cattle and sheep, which weighed as much as 42 pounds, Schlegel found necrotic passages, caused by boring, which were filled with distoma-brood; on the serous membranes of liver, spleen and lungs he found granular or fibrous, irregularly speck-like or worm-like deposits which were twisted, streaky, as long as half a finger, and which had yellow necrotic spots. In the parenchyma of the liver he found necrotic foci, up to hazelnut size, of a bloody brown-red or gray-yellow to greenish color, these contained living or dead distomas in various stages of development. Similar passages, which usually were bored in an oblique direction from the mucous to the serous membrane, were found in the wall of the small bowel, in that of the forestomachs, the mesentery and in the omentum; the greatly enlarged mediastinal, portal and bronchomediastinal lymph nodes contained foci up to pea size, yellowish green, caseous, or bright red; they contained distomas. Schlegel concludes that the frequent parasitic green nodules in the mesenteric and pulmonary lymph nodes are produced most frequently by strayed distomas and not by pentastoma larvæ.

According to v. Ratz, the nodules which are found in the liver of horses, millet-seed to hazelnut size, and calcified, are sometimes due to eggs of liver flukes (Olt found the remnants of young echinococci in such nodules, Mazzantini found embryos of filaria). Morot found distomas in tubercle-like nodules of the peritoneum, Joest in abscess-like nodules of the spermatic cord in a bull, and Cocu in the coagulated blood on the valves of the right heart.

In less severe cases of chronic distomatosis the liver may appear healthy from the outside, but if palpated, hard cords can be felt. On section these are found to be dilated bile ducts with thickened and rigid walls. Pressure causes dirty, yellowish-brown bile to exude, containing flukes in variable numbers. Similarly altered ducts can sometimes be seen through the capsule, especially on the posterior surface, as white firm nodulated cords.

In more severe cases the liver becomes enlarged as a result

of the chronic interstitial inflammation which appears later, and its consistency is at the same time firmer. The large bile ducts appear greatly dilated, their walls thickened and hard, and their inner surface is usually roughened by a deposition of salts (calcium phosphate and a little magnesium phosphate). In the lumen of the ducts greenish-brown, viscous bile and large numbers of flukes in various stages of development are found. In a single liver there may be 1,000 flukes or more. At a later stage there is shrinking of the liver, the organ becomes tough and the bile ducts appear as dilated tubes with calcified and thickened walls. Marked glandular proliferation of the mucous membrane of the bile ducts is almost always present (Schaper). As a result of repeated invasions various stages of the disease are often discoverable in a liver.

In all ruminants an unequal distribution of the changes can be observed, since the left lobe always shows a greater invasion. Particularly in light cases of distomatosis, a cholangioitis, with or without atrophy of the liver tissue, is found either exclusively or predominantly in the left lobe, while in severe cases an atrophic cirrhosis, which may even resemble cicatricial tissue with hyperplastic cirrhosis of the other lobes, may be observed. This peculiarity undoubtedly depends upon the anatomical arrangement which in some ways favors the immigration of the flukes. In severe cases there is usually ascites as well as general wasting and anemia.

The anatomical changes vary in the different species. In massive invasion in cattle a general although unequal proliferation of connective tissue is always found. As a result of this the liver increases up to threefold in weight, becomes firm and appears light gray in color. In sheep, on the other hand, the proliferation of connective tissue reaches only a medium degree even in severe and fatal cases of distomatosis, and the thickening of the wall of the bile ducts reaches from 1 to 3 mm. at the most.

Concerning the correlation between the number of Distomum hepaticum and the degree of the changes in the liver, we may conclude, according to the observations of Marek, that macroscopically only a cholangioitis is observed in cattle harboring from 116 to 244 flukes. In cattle harboring from 276 to 542 flukes a more or less pronounced proliferation of connective tissue and a shrinking of the liver tissue along the edges of the left lobe can be observed. In cattle harboring more than 550 flukes there is always an increase of from one and a half to three times in the size of the liver (in one cow with 1,660 flukes the liver weighed 14 kg.). In a sheep with 98 flukes only a cholangioitis was present, and in animals with more than 100 flukes in the winter months there was also an increase in the interstitial connective tissue.

The number of flukes (which can be accurately determined

only by a systematic dissection of the liver) varies greatly. Only a few, or up to 100 or more than 1,000 specimens may be found in the bile ducts (in the author's cases the highest number found in cattle was 1,660, in sheep 368; in Friedberger's cases in sheep 600; in the case of Dupray over 1,000), while a few may be found in the intestinal tract as far back as the colon. After the liver gets cold the active movements of the flukes cease, but when placed in physiological salt solution at body temperatures not later than 14 to 18 hours after the death of the host they will again become active.

Distomum lanceolatum causes changes similar to those caused by Distomum hepaticum only after massive invasion, but as a rule the process is inclined to be limited to the bile ducts and the chronic changes are not so severe. In spite of the fact that, according to Friedberger, their number may reach several thousand (in two of the author's cases in sheep 2,901 and 3,924 were counted), they may be easily overlooked or mistaken for blackish masses of bile. In cases of massive invasion they may be squeezed out of the crosssectioned bile ducts in sausage-like lumps. If only Distomum lanceolatum is present, disturbances of nutrition are absent (Friedberger, Neumann, Marek).

Symptoms. Visible disturbances of health seem to occur in cattle only when at least 250 large flukes are present, and in sheep when at least 50 large flukes are present. In young animals a smaller number of flukes may cause disturbances of health.

In acute cases, the period elapsing between infection and the appearance of symptoms is seventeen to twenty-one days. In chronic cases one to two months may elapse (Gerlach, Zürn, and others). The conditions for the development of the distomas being most favorable in summer and fall, the outbreaks of distomatosis, in acute cases, take place during the months of summer and fall, the ordinary chronic cases occur in the fall and winter months. The first symptoms of the disease can appear also in other seasons, in accordance with the fact that the invasion is possible at other periods.

In the majority of cases, **sheep** show no symptoms for a month or two after infection, and the first symptoms seen are slight fever, dulness, weakness and anorexia. Careful examination will show that the hepatic region is painful on pressure and that the area of dulness is increased. The posterior border may extend beyond the costal arch and can be palpated (Spinola). Anemia due to a reduction in the hemoglobin content and in the number of blood corpuscles may be demonstrated at an early stage and even in slight cases (Schaper).

In chronic cases, *usually about the end of the autumn*, there is edema. Simultaneously with the gradual paling of

the mucous membranes there is edematous swelling of the conjunctiva which forms a prominent ring around the cornea, and causes swelling of the eyelid. Doughy swellings make their appearance along the trachea, under the brisket, and on the abdominal wall. By this time the dulness and weakness are pronounced, the appetite is suppressed, rumination slow, the wool dry and brittle and easily pulled out, and it even comes out spontaneously at places thus making the fleece ragged. Large numbers of fluke eggs can be found with the microscope in the feces. By this time there is evidence of hydrothorax and ascites.

Disturbances of digestion are seldom absent. Particularly a permanent or periodical decline of appetite and a periodically light, seldom severe, diarrhea are observed. Also periodic attacks of fever are quite often observed. Icterus, on the other hand, is usually absent, or is present to only a slight degree, while the conjunctiva presents a light yellowish, grayish-white color, due to a shining through of the bloodless submucous tissue. Wester often found the liver enlarged and palpable on the right side behind the costal arch.

About the third month of infection, generally at the beginning of winter, the wasting becomes more and more pronounced and the edema and ascites are more evident. The edema of the tracheal region decreases during the night and reappears when the animals are at pasture during the day, while the edema of other parts tends to decrease a little during movement and to reappear with rest. In addition to edematous swelling, Mergel observed abscesses on the neck and below the flank which he believed were due to flukes.

Coincident with the increasing emaciation, symptoms of anemia and cachexia become more and more prominent. In some cases there is diarrhea, the feces containing large numbers of eggs. The milk becomes watery and less nourishing, so that the lambs are poorly developed and may even die. Finally, the animals die from complete exhaustion.

Spontaneous cures in a clinical sense are frequently observed, especially in well-nourished and also in older animals. Such animals recover toward the end of winter or the beginning of spring and gradually recuperate during the next grazing period, but remain carriers for a long time. Only seldom is the health of such animals impaired to a certain extent by the more or less extensive lesions in the liver which remain after the flukes have gradually been expelled. It is not improbable that many viable flukes remain in the liver of such animals but do not disturb the health of the animal while at pasture or on green feed, and produce manifest symptoms only during the next following winter. The occurrence of many cases of distomatosis also during dry years, in spite of the fact that in such years a

considerable invasion can hardly take place, can probably be explained in this way.

In **goats** *the symptoms are very similar to those seen in* sheep and diseased animals often abort, or their progeny are very weakly.

Fig. 90. Edema of the throat in distomatosis in sheep.

Cattle, buffaloes and **camels** present similar symptoms, but on account of their greater power of resistance the disease is as a rule, less severe. In these animals, edematous swellings along the course of the trachea and in the dewlap are more rarely seen, but there may be digestive disorders such as anorexia, diarrhea, and tympanites and, in severe cases, anemia and emaciation. A larger proportion of animals recover, and the disease may remain latent, although the infestation may be severe (Wiegel). *In exceptional cases, and especially among* young animals, death is due to utter exhaustion. According to Vaelzen and Pease, many buffaloes are slaughtered in the East Indies on account of distomatosis.

Very occasionally, the disease runs the same course in **solipeds** (ass) as in ruminants, wasting, anemia, dropsy, and in some cases jaundice being observed (Prietsch).

In rabbits, according to Braun, there are observed emaciation, ascites, collection of fluid in the pleural cavity, sometimes *edematous swelling, and icterus of the mucous* membrane. *On* the other hand, de Does and Sohns have reported a characteristic type of distomatosis in rabbits and guinea pigs. At first a paralysis of one of the hind legs, seldom of both, occurs, as a result of the formation of multilocular cavities with a connective-tissue capsule, which shows hemorrhages in some places and contains a dark brown fluid and a liver fluke. In

guinea pigs these cysts later appear under the skin as fluctuating knots the size of a pea. Such animals die as a result of decubital gangrene or from exhaustion. Upon postmortem similar cavities are found in the lumbar muscles in guinea pigs, and also in the lungs in rabbits and guinea pigs. At the beginning there is acute hepatitis resulting from the immigration of young flukes, 1-3 mm. long, which can also be found on the surface of the liver, resembling flocculi of exudate. In advanced cases the liver in guinea pigs may be entirely unchanged and free from flukes.

Course. Acute cases in which sheep die in seven to nine days after the onset of symptoms from acute hepatitis, are only rarely encountered (Bouvicini). In some cases, death is quite sudden, possibly owing to embolism of young flukes in the vessels of the brain (Gerlach) or to hemorrhage into the peri-

Fig. 91. Increase in the area of dullness of the liver in a cow with distomatosis. The dotted line represents the normal boundary of the liver, and the continuous line shows the boundary dislocated downward and backward and joining the lungs in front.

toneum following perforation of the capsule of the liver. With such exceptions the disease tends to become chronic. The course of the disease is divided by Gerlach and Zündel into four stages. (1) The stage of traumatic hepatitis which begins soon after infection, lasts four to thirteen weeks, and is characterized by slight symptoms of fever, digestive disorders, and enlargement and sensitiveness of the liver. (2) The stage of chlorosis. This sets in in the autumn, lasts six to twelve weeks, and is characterized by symptoms of anemia. (3) The stage of wasting as a rule sets in about four months after infection during the winter months. In this stage emaciation and dropsy are pronounced and deaths are frequent. (4) The migration of the flukes occurs in the spring, about May or June (Gerlach). During this stage, symptoms disappear and there may be temporary or permanent improvement. The investigations of Friedberger and Schaper have shown that this division into

stages is applicable to a certain extent in cases where there are extensive outbreaks because there are a number of animals severely and simultaneously affected. Such outbreaks generally occur in the summer and autumn.

In most cases the stages cannot be marked off from each other and the series of symptoms described above cannot be followed out. The eggs of the parasites may be passed out and the parasites themselves migrate at any time. There is great variation in the duration of the disease. In some cases where the infestation is severe, it may last only three months, and in another case it may extend to a year or more.

Diagnosis. Obvious symptoms are seen in advanced cases only, and more or less closely resemble those due to other causes (tapeworms, gastric strongyles, etc.). The edematous infiltration cannot be considered as characteristic of liver rot. As a rule, no difficulty is experienced in practice, in arriving at a diagnosis when there is a dead animal available for examination. During life a certain diagnosis can be based upon the discovery of the eggs of flukes in the feces. It must be remembered that in the early stages of the disease there may be no eggs in the feces and in such cases, enlargement and sensitiveness of the liver should arouse suspicion as to the existence of liver rot. This suspicion may be supported by a history indicating the likelihood of infection. By the use of the agitation method in the microscopic examination of the feces (see page 321), and counting the fluke eggs while systematically searching all parts of 10 specimens for the cow and 5 specimens for the sheep and goat, one can, after some practice, determine approximately the number of flukes in the bile ducts, and accordingly the degree of invasion, and thus come to a conclusion as to whether the symptoms are wholly or partly due to flukes. According to the figures of Marek and Pataki, which agree very closely with the postmortem findings in the Budapest Clinic, 6 eggs of Distomum hepaticum in 10 preparations from a specimen of feces represent on an average 100 mature liver flukes in cattle, while in sheep and goats 17.5 eggs in 5 preparations represent 100 mature liver flukes. Under the same conditions, 2 eggs of Distomum lanceolatum represent 100 mature flukes in sheep. However, in this case the relation is much less constant, and the rather small eggs are much harder to find, as a result of which the microscopic examination may be negative, especially when one has not had sufficient practice, in spite of the fact that eggs may be present in large numbers.

The complement-fixation test, for which a filtered saline extract of liver flukes is used, and which Weinberg reports quite reliable, has only a subordinate practical significance as compared with the microscopic detection of the eggs.

Prognosis. If the symptoms are pronounced, prognosis is unfavorable. The symptoms may abate from time to time, but there is scarcely ever a complete recovery. Spontaneous improvement and recovery in less severely affected animals are not infrequent, but even severely affected animals can be saved by proper treatment. Young and weakly animals are generally affected more seriously than older and more robust ones, and cattle survive infection better than sheep.

Treatment. According to the most recent experimental results, a cure of distomatosis can be effected by destroying the flukes with extract of male fern. This drug, which even in small quantities destroys the flukes, is absorbed from the intestinal tract and is carried to the liver with the blood, from which it is taken up by the flukes while sucking blood from the walls of the bile ducts. According to the systematic experiments of Railliet, Moussu and Henry, which were carried out following preliminary experiments by Grassi and Calandruccio, Perroncito and Alessandrini, extract of male fern containing from 15% to 25% of the active principle of filicis (to be determined titrimetrically and to be checked periodically) is suitable for this purpose. Upon the recommendation of Moussu, this drug is administered on 5 or 6 consecutive days to sheep and goats at the rate of 1 gm. for 6 kg. body weight (more recently a single dose of 8 gm. extract of male fern is recommended), and to cattle 1 gm. for 10 kg. body weight together with from 2 to 5 times the amount of neutral oil. The so-called fasciolin, which is extract of male fern mixed with extract of mussena bark, is essentially also an extract of male fern. (Dosage, sheep up to 1 year old, 3 cc.; over 1 year old, 5 cc.; cattle, 15 to 20 cc. with 4 times that amount of neutral oil on five consecutive days.) Most suitable under all conditions is distol, which retains for years its unvarying content of readily absorbable, purified filicis substances dissolved in lipoid-soluble material in gelatin capsules (Marek). Sheep and goats up to 20 kg. body weight receive 1 gm.; from 20 to 30 kg., 2 gm.; 30-35 kg., 2.5 gm.; 35-45 kg., 3 gm.; 45-50 kg., 3.5 gm.; 50-60 kg., 4 gm.; or 0.07 to 0.09 gm. per kilogram of body weight; in each case on two consecutive days, or one-half the daily dose on four consecutive days. The dose for cattle and bisons up to 120 kg. is 3 gm., and for every additional 50 kg. body weight, 1.5 gm. more (for animals of 170-220 kg., therefore, 6 gm.), on four consecutive days, or, better, 0.03 to 0.04 gm. per kilogram of body weight (the animals should be weighed or their weights carefully judged).

According to other experiments of Marek, liver flukes in sheep and goats can also be destroyed with kamala administered in pills. Sheep over 1 year old received 7.5 gm. daily on two consecutive days; weak animals or animals suffering with diarrhea, 3 gm. on five consecutive days; sheep less than 1 year

old received 4 to 5 gm. Cattle will not stand kamala in amounts sufficient to destroy the liver flukes (it causes exhausting and even fatal diarrhea). Parasitin, calbazin and calbazan are proprietary drugs containing kamala and are found on the market in Hungary. Kamala has recently been shown to be entirely unreliable, which is probably due to the fact that the drug has been improperly stored and kept too long.

Besides medicinal treatment, the animal should be properly fed with good hay, grain, bran, oil cake, etc.

Administration of Extract of Male Fern. The extract should be kept in a cool, dark place and in well-stoppered bottles. Preparatory to administration the drug is well shaken up and the required amonnt, computed on the basis of the number and

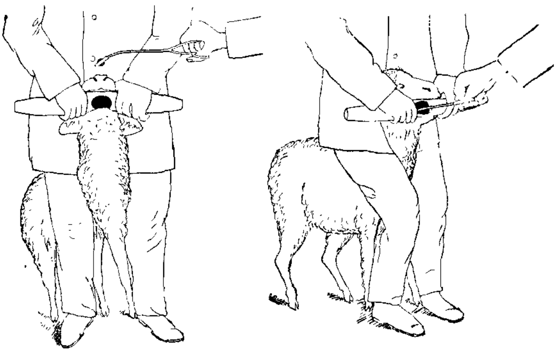


Fig. 92. Fig. 93.
Convenient administration of pills or capsules in sheep for mass treatment. Fig. 92, opening of the mouth and restraint of the animal. Fig. 93, introduction of the pill or capsule into the pharynx with slightly curved forceps.

the body weight of the animals to be treated, is thoroughly shaken with the necessary amount of neutral oil, and the proper amouut of the mixture is administered in the usual manner by means of a narrow-necked bottle. Sheep and goats are best dosed with a fairly stiff rubber tube that has a funnel attached to one end. The tube is introduced into the esophagus for a distance of at least 20 to 30 cm., to prevent any of the drug from getting into the trachea. In these animals, extract of male fern may also be administered in gelatin capsules (Strouhal). It is furthermore recommended that the drug be administered on an empty stomach.

Administration of Distol. This drug, which can be obtained already prepared is administered to cattle in the usual manner with the hand. An assistant restrains the animal by catching it in the nostrils with one hand and opens the mouth by catching the animal on the lower jaw with the other. The operator then draws the tongue of the animal forward and places the capsule well back upon the base of the tongue. In case many or young animals must be treated, it is much more convenient and preferable to use forceps lightly curved at the end and 30 to 35 cms. long, similar to those used in sheep (Figs. 92 and 93).

In treating sheep and goats an assistant catches the head of the animal between the legs, with the head turned toward the light, places a thin, smooth board 30 cm. long with a hole 3 to 4 cm. wide in the middle into the mouth of the animal, and with it presses down the point of the tongue. The capsule is now taken up with the forceps, carried along the hard palate and up to the soft palate and released. The forceps are immediately withdrawn and the board is also quickly removed from the mouth so that the animal may swallow undisturbed. Should the capsule be allowed to drop out of the mouth, it is again administered in the same manner. Should the capsule happen to burst in the mouth or in the pharynx, this will not interfere with the curative effect if the head of the animal is properly held up so that the drug cannot flow out of the mouth. The treated animals should be kept separate from those not yet treated so as to prevent the possibility of administering a double dose. If only a few sheep or goats are to be treated, the method of simply administering it with the hand may be used. It is not necessary to give the drug upon an empty stomach, but it may be given at any time.

In animals that are already greatly exhausted or show a total loss of appetite or diarrhea, the extract of male fern may also be administered intravenously in alkaline solution in single doses of 0.006 gm. per kilogram of body weight. Sheep and goats should receive two doses, and cattle at least four doses at intervals of one day.

Administration of Kamala. This drug can be administered most conveniently in pill form. The drug is made into a dough with double the amount of flour or linseed meal or powdered althea root and double the amount of water. For weak animals or those suffering with diarrhea a little powdered oak bark is mixed with it. The dough is then formed into one or several pills and administered in the same manner as capsules. For mass treatment it is best to mix the required amount of flour and water into a dough and have it ready at hand. Just prior to dosing, the amount required for each animal (for sheep a piece the size of a walnut) is mixed with the required amount of measured kamala and formed into a pill.

Direct Effect of the Fluke-Killing Drugs Upon the Animal. Since the drugs are administered in relatively large and repeated doses so that the active substances may reach the bile duct in sufficient quantities, it is not surprising that they should affect the host more or less. In some animals extract of male fern produces for a period of several days a decrease of appetite or exceptionally total loss of appetite, slight diarrhea, debility, seldom lethargy or paralysis-like weakness. Even a fatal intoxication of the animal may result if the prescribed single dose is exceeded, as in the case of a steer weighing 500 kg. which received three doses of 50, 60 and 70 gm. extract of male fern.

In accordance with its greater content of filicis substances, the administration of distol is followed, in cattle as a rule, and in sheep and goats in one-half of the cases and for a period of one-half to three days, by a decrease in appetite, light to moderate diarrhea, and debility. The drug has no influence on pregnancy. Animals that are already cachectic sometimes become exhausted to such a degree that they die (Reisinger and others). In a large flock of sheep amaurosis was observed in 8 animals after 2 doses of 3 gm. each were given. In 4 of the animals the disturbance of vision disappeared after several days.

Kamala always produces severe disturbances of health (debility, weakness, depressed appetite, diarrhea) for a period of at least 3 to 5 days, and in flocks of weak animals it may cause heavy losses. It may also lead to a permanent cessation of the milk flow, and in suckling animals may also produce a severe or even dangerous diarrhea, since the drug is excreted with the milk.

Result of the Treatment Upon the Liver Flukes and Upon Distomatosis. Under the influence of the administered drugs, the liver flukes begin to succumb by the end of the first day of treatment, and all may have died at the end of the following day, although as a rule the last do not die until two days (distol) or 3 to 8 days (extract of male fern, kamala) following the completion of the treatment. The killed liver flukes (Fig. 94) are extended, become limp, and are stained yellowish brown or light green by the bile. Soon after they appear as though frayed; many of them are converted into the form of a cord by a curling up of the edges, or into a veil-like structure showing remnants of the cuticle and branches of the intestinal tract. Now and then, flukes are found that have died only in part, particularly in the posterior half of the body (Fig. 94b), but later succumb when at least one-third of the body has perished, unless the anterior part of the body was first attacked, in which case a less extensive injury may bring about the death of the parasite. After the death of the parasites they are carried away by the bile, or degenerate within the bile ducts, so that after the above-

mentioned time no flukes are found in the bile duct. On the other hand, the expulsion of the eggs of the flukes may continue for two weeks or longer, or even six weeks, although the majority ot the eggs have left the body of the host within two weeks.

The effectiveness of extract of male fern in particular is influenced especially by its content of active substances, which vary according to the origin of the fern and the method of preparation and preservation of the extract. (In France extracts with a very high percentage, 24 to 25 per cent., of filicis substances are produced.) For this reason the results obtained with the drugs are quite variable. While with the French extract Railliet, Moussu and Henry were able to destroy all flukes in 3 out of 7 sheep (57%) and achieved a marked reduction in the number of flukes in the other animals, Marek could destroy

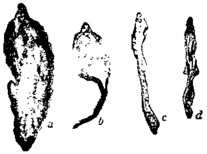


Fig. 94. Dead Liver Flukes. a, Degenerated liver fluke; b, liver fluke dead and rolled up in the posterior end; c, shrivelled and veil-like transparent liver fluke; d, cord-like, shrivelled liver fluke.

all the flukes with extracts of various origin in only 25% of 24 sheep, while in another 29% of the animals not even a reduction in the number of parasites could be achieved. In cattle the experiments were entirely negative. In Strouhal's cases, in 3,000 sheep, the extract seems to have been entirely ineffective. For this reason only extracts with a known high content of filicis substances should be used.

Following internal administration of distol, Marek observed the destruction of all flukes in 82% of the sheep and goats (more recently in 100%) and in 72% of the cattle. In the remaining small percentage only a few flukes escaped destruction, and these, because of the small number, could no longer injure the health of the animal. Similar results were also recorded by Reisinger, Hetzel, Schermer, de Blieck and Boudet and others.

In the experiments of Marek the action of kamala in sheep coincided with that of distol, while Moussu and Schermer found it entirely unreliable.

The symptoms of the disease disappear very rapidly even in very sick animals. After the first to the fourth day the appetite gradually returns; diarrhea, if any was present, disappears, the animals become more lively, their condition improves

rapidly, and in from 2 to 3 weeks the normal color of the mucous membranes may have returned. On the other hand, the drug has no effect upon the anatomical changes in the liver tissue, although the further progress of the process is arrested in all cases where the flukes have been destroyed. This explains the observation that cattle treated in a very advanced stage of severe illenss cannot be fully restored, on account of the severe cirrhosis of the liver that has already occurred in spite of the fact that all flukes have been expelled, whereas otherwise they would have fully recovered.

The most appropriate time for treatment is usually at the end of autumn or the beginning of winter, at which time advanced changes in the liver of cattle have not yet occurred and the animals are still strong, and hence do not suffer so much from the drastic treatment. Cattle in less advanced stages of the disease may be treated at any season of the year, and the same applies to sheep and goats, without regard to the origin of the infestation, provided they have not already become too weak.

Before beginning the treatment for distomatosis one should seek to determine whether or not other diseases (worm disease of the lungs, stomach and intestine, emaciation and dydremia resulting from insufficient food) endanger the life of the animal, while the invasion with liver flukes may be only of a light character and play only a subordinate rôle.

Undesirable harm to sheep, other than the direct result of the treatment, may be produced by less apparent or latent infectious diseases, as the drastic treatment may cause these to become acute and spread rapidly, leading to the death of many animals. In this connection there should be mentioned, according to Wiemann (see Vol. 1), particularly a streptomycosis, and according to Carre and Bigoteau (see Vol. 1), pseudotuberculosis, also the chronic types of hemorrhagic septicemia (see Vol. 1), as well as lung-worm disease. Streptomycosis was determined experimentally and also observed in several flocks. In case such latent infectious diseases are suspected, the sheep should be placed in another barn and only a few animals or small groups of animals, treated consecutively. If in the course of several days no symptoms of an infectious disease have appeared, the remaining animals may be treated.

Other Methods of Treatment. Remedies formerly used are benzine (sheep 5 to 6 gm., cattle 60 to 120 gm., for 4 to 6 days), naphthalin (sheep 0.7 to 1 gm. twice daily for one week), oil of turpentine (sheep by the tablespoonful), carbon bisulphide (according to Floris, 10 to 15 gm. for adult animals in gelatin capsules for 4 to 6 weeks).

All drugs which destroy the common liver flukes are without effect upon Distomum lanceolatum. This, however, is without significance, since, alone, this parasite only seldom causes disturbance of health.

Prophylaxis. A very effective means of prevention is to avoid moist, swampy pastures, although it can only seldom be carried out. But little success is also promised by feeding 7 to 8 gm. of sodium chloride with the hay or giving it in drinking water up to 0.5% each time before the animals are taken to pasture. Only a decrease in the degree of invasion can be expected by allowing the animals to graze over a larger territory so that they need not crop the grass so closely. According to von Linden, copper salt licks are supposed to protect the animals against invasion with liver flukes (see lungworm disease).

Drainage is very effective for destroying the infectivity of the pastures. However, this frequently meets with local or economic difficulties. The application of lime to the infested pastures and meadows from spring to the end of September in the form of 800 to 1,000 kg. lime water (made up of 3 kg. fresh air-slaked lime and 1,000 liters of water) per hectare (2½ acres) is more readily accomplished and destroys not only the Mirazidæ but also the Linnaeae. The application, which is harmless to animals, should be frequently repeated during wet years and following overflows, since the effectiveness of calcium hydroxide is soon destroyed by the conversion of this compound into calcium carbonate. French veterinarians also recommend that the pastures be uniformly covered with iron sulphate (300 to 500 kg. per hectare), or sprinkled with a 0.1% solution of iron sulphate (800 to 1,000 liters per hectare).

To prevent a new infestation of the pastures, Schaper recommends that infested animals be kept in a barn or placed in lots on hard, dry ground, while the manure should be applied only to well-drained fields. Mixing the manure of sick animals with lime or iron sulphate is less practical and less reliable. Destroying the infested livers or boiling them before feeding them to dogs—since dogs will otherwise expel the eggs of liver flukes in a viable form with the feces—will also help to check the disease. Collecting the Limnaeae or destroying them by allowing geese and ducks to graze on the pastures should also be considered. According to Galli-Valerio, Aulastoma gulo, which belongs to the true horse fluke, should also be considered, as this creature will destroy the Limnaeae by sucking on them.

Total eradication of the liver flukes, or at least a decided decrease in the infectivity of the pasture, could be attempted by systematically expelling all flukes from the animals for several years at the time when the animals are placed in winter quarters, and, if necessary, also a few weeks before they are allowed to go to pasture again in the following spring. The manure of the animals should be collected for two weeks following each treatment and should be placed only on well-drained fields. Each newly introduced animal of a susceptible species should be given a course of treatment if a reliable microscopic examination of the feces does not show it to be free from infestation.

Other Distomes in the Liver. · Opisthorchis felineus (Distomum felineum) occurs occasionally in the livers of dogs and cats: the infection is probably brought about through the mediation of certain fish in which the embryo develops; the eggs are provided with a polar cap; at the opposite pole they carry a small protuberance; their length is 26-30 μ. In the case of a massive infection, the biliary passages are dilated and chronic hepatitis develops. Opisthorchis novorca (D. conjunctum) was found, by Lewis, in cysts and pus foci in the livers of dogs in India. Metorchis truncatus (Amphistomum truncatum) occurs in the livers of cats and dogs. Metorchis albidus (Distomum albidum) lives in the livers of cats, rarely in dogs. Clonorchis (Opisthorchis) sinensis is parasitic in the livers of dogs and cats; its eggs resemble those of the Opisthorchis. Amphistomum explanatum, which belongs to the family of Amphistomides of the Trematodes, may cause massive infection in cattle and buffaloes in Cochin-China, and then gives rise to dilatation of the biliary passages associated with a whitish yellow discoloration of the liver.

(c) Cysticercosis of the Liver.

(Hepatitis cysticercosa.)

Cysticercosis of the liver is an acute or chronic hepatitis caused by the Cysticercus tenuicollis or C. pisiformis with secondary peritonitis.

Occurrence. Cysticercosis of the liver is seen principally in lambs, young pigs and rabbits or hares. It was also seen in a doe, in calves (Falk) and in the cow (Pütz). In the first mentioned species it usually occurs as a local disease and causes heavy losses (Kuehnau, Brusaferro, Avéradère, Moussu, Duerbeck, Seiler). Almost without exception animals between the ages of a few weeks to several months are affected.

Etiology. In cattle and pigs the disease is caused by the Cysticercus tenuicollis, which is the cystic stage of the Tænia marginata which inhabits the intestine of the dog (see page 347). In rabbits and hares the cause is the Cysticercus pisiformis, the cystic stage of the Tænia serrata also an inhabitant of the intestine of the dog (see page 347).

Infection occurs naturally through the ingestion of feces of dogs harboring these parasites. The proglottides or the contained eggs are rarely dropped directly into feeding- or drinking-troughs. As a rule, the feces are dropped about on the ground or in the neighborhood of drinking places and thus contaminate the food or water. Pigs may ingest them while rooting.

Susceptibility. It cannot be determined whether there is any difference of susceptibility among animals that harbor the Cysticercus tenuicollis. The varying incidence of the disease

in the different species may be due to differences of surroundings. Age has some influence upon the susceptibility, since young animals are attacked more frequently and more severely. The fact that animals are affected at the age of a few weeks, shows that within that time the opportunity offers of ingesting the eggs of tapeworms. Young pigs may also ingest the eggs while rooting.

Pathogenesis. The embryos of the Tænia marginata and T. serrata are liberated from the eggs by the acid stomach contents, and reaching the intestine bore through the wall and enter the portal veins by which they are carried to the branches of the portal vein in the liver. They have been found there four hours after the ingestion of the eggs (Hoffmann). Some embryos may pass through the capillaries into the hepatic veins, and thus be carried to the lungs or other organs. After their arrest in the blood vessels they bore their way out, and penetrating the liver tissue, make their way to the surface leaving tracks behind them. Finally, some of them perforate the capsule of the liver and reaching the peritoneal cavity develop further in the omentum, mesentery, or peritoneum. Those that remain under the capsule of the liver also undergo processes of development. The same thing happens to the embryo arrested in the vessels of the lungs.

A severe invasion causes a condition similar to that produced by a heavy infestation with flukes (see page 479). There is an acute hepatitis often associated with hemorrhage and a localized or wide-spread peritonitis. The rupture of large vessels may cause fatal hemorrhage. If the animals survive, the acute hepatitis is followed by an interstitial inflammation, causing cirrhosis. In some cases the embryos in the lungs set up broncho-pneumonia and even pleurisy.

Anatomical Changes. In the acute stage the liver is more or less enlarged, although there may be no enlargement in some cases. Its serous membrane is dull and may be covered with a hoar-like fibrinous layer. In many cases the surface of the organ appears nodular and the apex of each nodule shows a minute opening. The liver itself has a speckled, mosaic-like appearance (mosaic liver or trout liver) on account of the variation of color of the lobules. Some of these are black or blackish-red and others are pale red or grayish-brown. The darkly colored lobules are enlarged. The consistency of the organ is soft, and the tissue is easily broken down. Close examination sometimes shows within the dark-colored lobules yellow points or transparent acephalic cysts measuring three to four mm. in length, by one to two mm. broad. In the later stages there can be seen burrows in the liver tissue which at first were filled with blood and afterwards become yellowish-gray in color as a result of degeneration of the injured tissue,

and the outpouring of an exudate. These burrows are sometimes sinous and sometimes wider at one end.

Microscopic examination reveals the presence of cysticerci in the little pools of blood and cellular infiltration in the neighborhood and in the interlobular connective tissue, especially at the angles of the lobules. Finally there is compression and partial degeneration of the liver tissue.

Not rarely there is acute peritonitis or there may be liquid blood in the peritoneum. In this blood may be seen numbers of very minute pale-colored bodies (young cysticerci).

In chronic cases there is more or less chronic interstitial hepatitis and shrinkage of the new connective tissue.

Symptoms. The symptoms of acute cysticercosis of the liver set in a few days after infection. This has been proved by the feeding experiments of Küchenmeister, Leisering, Leuckart, Baillet and Railliet. A kid infected experimentally by Railliet became seriously ill nine days later. In young pigs the course of the disease is sometimes very acute, animals dropping dead without apparent cause. In other cases the symptoms are depression, staggering gait, and marked dullness. Death may be sudden.

As a rule the evolution of the disease is more gradual. Dullness, great weakness, inappetence, and thirst may be observed, and in the later stages acute peritonitis with fever, distension of the abdomen and sensitiveness of the abdominal wall. In such cases there is rapid emaciation. Death may occur within a few days or the animals may live for some weeks until there is marked anemia and debility.

Chronic Cysticercosis. This is the form of the disease usually seen in the rabbit, but it occurs also in other species of animals. The symptoms are chronic digestive disorders which cause some loss of condition.

Diagnosis. The specific nature of the disease is best shown by the demonstration of young cysticerci in liquid withdrawn from the peritoneum. As a rule a diagnosis is only possible post mortem, although a strong suspicion as to the nature of the disease may be raised if there are a number of young animals showing the symptoms described, and especially if the surrounding circumstances are favorable to infection. In very recent cases or when there are no burrows in the liver, tissue diagnosis may be attended with difficulty even at the autopsy, especially if no microscopic examinations are made. In such cases other forms of acute hepatitis and other infectious diseases (swine erysipelas, hemorrhagic septicemia) have to be excluded. Acute distomatosis is more easy to diagnose because of the presence of young distomes which measure at least one to three mm., either in the burrows in the liver tissue or in the peritoneum. Weinberg found the complement-fixation test to be reliable for diagnosis.

Prognosis. Prognosis depends entirely upon the severity of the infestation, but in acute cases it is unfavorable. Avéradère records a mortality of 38 per cent in an outbreak among lambs. Chronic cases are fatal far more rarely, especially in adult animals, and as a rule there are no symptoms in these cases.

Treatment and Prophylaxis. Treatment must depend upon the nature of the case. As a prophylactic measure all dogs in the neighborhood of the animals and especially sheep dogs, should undergo a course of treatment for tapeworms two or three times a year or, if possible, should be kept quite away from the animals.

(d) Coccidiosis of the Liver. Coccidiosis hepatis.

Coccidiosis of the liver is an enzootic disease among rabbits, affecting the liver and the bile ducts and is caused by the Coccidium oviforme.

Etiology. The **Coccidium oviforme** Leuckart (Eimeria Stiedae) has a double outer membrane with one pole slightly flattened and measuring 30 to 50 μ in length, and 14 to 28 μ in breadth. (For the development of coccidia, see page 404.)

Natural Infection. The natural method of infection is by the ingestion of food or water contaminated with the feces of rabbits that are affected with the disease or that have coccidia in their livers. Young animals are especially susceptible, and in them the disease is apt to be severe. Adult animals, on the other hand, are affected less seriously, and as a rule, show no symptoms. Such animals are capable of infecting young animals by means of their feces.

Pathogenesis. The sporozoites are liberated by the covering of the oocyst being dissolved, and then probably reach the biliary passages through the bile duct, invade the epithelia of the mucous membrane and multiply there by sporogony. They cause proliferation of the epithelia of the bile duct, connective tissue proliferation in the immediate neighboring tissue, giant cells in the proliferated wall of the bile duct which has become necrotic in places, and finally changes similar to cirrhosis of the liver. The oocysts, which are formed in the meanwhile, are transported into the intestine with the bile, and outside with the feces; they mediate the spread of the affection in the rabbits.

Anatomical Changes. The lesions vary in size up to a pea or even a hazelnut, and take the form of whitish-yellow nodules that are somewhat prominent and contain a creamy or caseous material. There is a connective tissue capsule. Coccidia are found in enormous numbers in the content of the nodules.

Occasionally coccidiosis of the liver is found in other mammalians, but it does not appear to produce any appreciable clinical symptoms. Johne demonstrated coccidia in cavities, as large as an apple, which he found in the liver of a hog. Perroncito & Rivolta found coccidia in an abscess of apple size, in the liver of a dog; in Bornhauser's case in a dog the coccidiosis gave rise only to proliferation of the epithelial cells of the bile duct and stenosis of the biliary passages. Chierici found, in a cat affected with liver coccidiosis, the liver somewhat enlarged, the biliary passages dilated and their walls thickened.

Symptoms. The symptoms are disturbance of nutrition, emaciation, weakness and, later, staggering gait, inappetence and eventually jaundice of the mucous membranes. In the last stages one observes diarrhea which is very persistent, a discharge from the nose, and finally convulsions. Young animals usually die in about two to three months, and whole warrens are often wiped out.

In young chicks coccidiosis of the liver, also known as sleeping sickness, occurs enzootically and manifests itself in sleepiness. It always terminates fatally.

Diagnosis. It is quite easy to arrive at a diagnosis during life by the demonstration of coccidia in the feces by means of the microscope. The feces contain fewer coccidia than in cases of intestinal coccidiosis. The two conditions are often observed in the same animal.

Treatment and Prophylaxis. Treatment is of no avail and efforts must be directed principally towards prevention. In this connection attention must be paid to the separation of the young animals from the adults as soon as possible, all visibly diseased animals must be destroyed, hutches must be disinfected, kept dry aud well ventilated. The hay racks should be made of wire fencing, and the feed boxes should be provided with slats or with wiring, to prevent the animals from crawling into them.

Other Animal Parasites Found in the Liver. The following parasites are sometimes clinically important as occurring in the liver:

1. The larvæ of **sclerostomes.** These reach the liver by way of the portal blood and make burrows in the liver tissue in a similar manner to cysticerci (Colucci, Mégnin, Schlegel). Schlegel repeatedly saw cases of chronic hepatitis which clinically resembled Schweinsberg disease, (see page 451), in which the liver was enlarged two to three times, and showed small nodules varying in size up to a pin's head, especially close under the capsule. In these cases there were also narrow tortuous burrows in the liver tissue of a red or reddish yellow color. The contents of the nodules and burrows were either caseous or calcified, but the larvæ of sclerostomes could be demonstrated.

2. **Eustrongylus gigas.** This parasite was found by Lissizin in the liver of a dog which had had convulsions for three days.

3. **Linguatula denticulata** (Pentastomum denticulatum) was found in large numbers in the liver of a goat that had shown symptoms of debility for a long time. The same parasite was found in the

liver of an otherwise apparently healthy ox. The capsule of the liver showed large numbers of openings measuring about 3 mm., and leading into cavities each of which contained a larval linguatula. Similar cavities were visible in the deeper layers of the liver.

4. Occasionally **ascarides** (Ascaris megalocephala, A. suilla) wander out of the intestine into the large bile-ducts in the horse and pig and may cause symptoms of colic or epileptiform seizures (Röll, Ortmann). In cats the embryos of the Ollulanus tricuspis which normally inhabits the stomach are sometimes found in the liver.

5. **Protozoal Hepatitis of the Pigeon** (Caseous hepatitis of the pigeon). Young pigeons sometimes die suddenly as a result of multiple caseous lesions in the liver without having shown any symptoms. In such cases the liver is enlarged and contains large numbers of grayish-yellow dry caseous foci. The foci vary in size from a millet seed to a hazelnut and are irregularly rounded in shape. Sero-fibrinous peritonitis is also present. The disease was first described by Rivolta in 1878 and later by Jowett (1907). Both authors found protozoa in the liver lesions provided with one or two flagella. This parasite was named the Cercomonas hepatis by Rivolta. In 1910 two cases were studied carefully by v. Ratz who found that the parasites were provided with three flagella and came to the conclusion that the parasite was the Trichomouas columbæ which usually inhabits the iutestine or respiratory organs of the pigeon. Under some circumstances which are not known they make their way ont of the intestine and penetrate the liver, there sitting up nodular necrotic lesions.

He believes that the parasite found by Rivolta and Jowett was the Trichomonas (v. Rátz, Közl., 1910, VIII, 184).

Martin & Daille found, in fatty livers of geese, cavities, up to walnut-size and similar to echinococci; they contained a wine-yellow, syrupy, gelatiuous substance which soon acquired the appearance of pus, when exposed to the air, and contained numerous blastomycetes, 5-31 μ in size, consisting of membrane and granular protoplasm, and frequently in the process of budding. They suggest the name of Cryptococcus anseris for them.

Section VII.

DISEASES OF THE PANCREAS.

References to diseases of the pancreas in the domestic animals are very scanty in literature. This is in part due to the fact that clinical investigation of the gland is impossible in the case of herbivora and possible to a slight extent only in the carnivora. The special methods of investigation that have been employed to obtain exact information regarding the digestive phenomena have not been undertaken from a veterinary point of view.

Symptoms in General. The fact that there may be no digestive symptoms in a case where there is a disturbance of function of the pancreas is due to the circumstance that to a certain extent the pancreatic juice can be replaced by the other digestive juices and by the intestinal bacteria. Disturbances in the pancreatic function become manifest through insufficient digestion of fats and, in consequence, by fatty stools (steatorrhea) in carnivora, the feces of which then show a gray discoloration and a peculiar fatty gloss; according to Mueller, the proportion of free fatty acids and of soap is much reduced. The incomplete assimilation of the proteins of the food may also produce azotorrhea, that is, the appearance of visible remnants of meat with stainable nuclei. On the other hand the sugar metabolism is deranged, with the resulting production of diabetes mellitus both in experimental animals and in natural cases of disease of the pancreas.

In certain diseases the pancreas can be palpated through the relaxed abdominal wall and in such cases valuable information may be gained, especially in carnivora. Nevertheless it is extremely difficult to differentiate between pancreatic lesions and tumors of the pylorus or enlarged portal lymphatic glands. This difficulty is also present in cases where the enlargement of the gland has caused jaundice or ascites by pressure on the bile ducts or portal vein.

The Cammidge reaction is often recommended for testing the function of the pancreas; it was carried out with positive result by Preller in a horse with pancreatic diabetes, and its positive outcome in general is claimed to denote disease of the pancreas. In observations of the authors this has not been confirmed in so far as a positive result was obtained in a case of intestinal catarrh, while the reaction was

Schmidt's nucleus test to demonstrate the absence of the capacity of the pancreas to digest the nuclei of the cells can easily be carried out in the dog. For this purpose pieces of muscle tissue 0.2 to 0.5 cm. long are hardened in alcohol and then washed in running water for one-half to 1 hour. They are then sewed or tied up in a piece of gauze and the animal allowed to swallow them. The pieces can easily be found again in the feces. If nuclei can still be demonstrated in the recovered

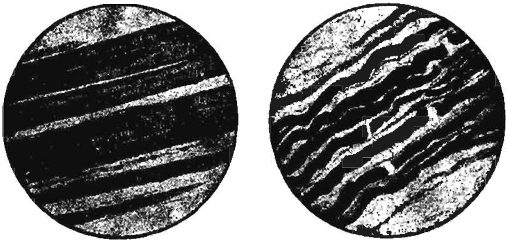


Fig. 95. Schmidt's cell nucleus test to test the activity of the pancreas. Left, positive in an 8-months-old dog with advanced aplasia of the pancreas (nucleus of sarcolemma and cross striation retained in the ingested pieces of muscle tissue): right, negative in a healthy dog (nuclei and cross striation absent, muscle fibers homogenoous).

muscle tissue by staining with hematoxylin it indicates the absence of trypsin. More recently Fronzig recommended the use of washed and dried nucleated red blood corpuscles of the frog or birds, which are simply mixed with the food.

The adrenalin mydriasis test with dogs having no pancreas was always positive. Three drops of 1-1,000 solution of adrenalin, suprarenin or tonogen are twice instilled into the conjunctival sac at intervals of five minutes. Thereupon, in cases of severe and extensive affections of the pancreas, dilatation of the pupils takes place (absence of the inhibitory action of the internal secretions of the pancreas upon the sympathetic nervous system).

Mégnin & Nocard described a **catarrh of the pancreatic duct.** The duct was completely occluded by a catarrhal secre-

tion with the result that there was a production of connective tissue, subsequent shrinkage of which led to atrophy of the gland parenchyma. Catarrh of the bile ducts and chronic interstitial hepatitis were present at the same time. During life the symptoms shown by the horse were: jaundice, partial loss of appetite, languor, unsteady gait and emaciation. The feces were dry and pale in color. In two months the animal was in a state of complete collapse.

Chronic Inflammation of the Pancreas. This was observed by Siedamgrotzky in a horse, the animal showing loss of hair, edema, leucocythemia and debility. The stroma of the pancreas was greatly increased in amount, and the gland tissue reduced, and the ducts were obstructed with viscid mucus and calcified flakes. (Kitt found a similar lesion in a horse.) In a case recorded by Wheatley, there was extensive cirrhosis of the gland which weighed sixteen pounds, the ducts were dilated and filled with material resembling albumen. The horse had shown gradual wasting, edema and excoriation of the skin at places.

Suppuration of the pancreas has been set up in conjunction with contagious rhinitis of horses (Reimers), also by the presence of foreign bodies in Wirsung's canal. In a case recorded by Goubaux in the horse, the foreign body was a straw stalk, and in the dog, Bruckmüller records the presence of a needle in the canal. Schättler found a piece of wire in a horse. The symptoms presented were loss of appetite, emaciation, occasional attacks of colic and susceptibility to pressure in the hypogastric region.

Nodular fat-necrosis of the pancreas occurs in absolutely normal lobules in fattened pigs (Marek, Rónai). It also occurs exceptionally in the dog and horse. No symptoms are produced and in cases where they cause sudden death they may be associated with multiple hemorrhages of the gland. Hemorrhages in the pancreas and surrounding tissues were found in two cases by Prettner and in one case by Mettam, in dogs which had shown symptoms of vomiting, abdominal pain and sudden collapse. Prettner is inclined to think that the condition was due to an infection from the intestine. In Mettam's case the gland was enlarged to a considerable extent and beset with necrotic foci. In one case similar lesions were found in a horse that had died from acute enteritis of the small intestine and peritonitis. The cause of necrosis of the fatty tissue has not been elucidated fully, but it is probably a question of the action of pancreatic ferment, possibly also due to hemorrhages.

Atrophy of the pancreas was found by Liénaux, Eber and Sendrail in dogs dead of diabetes mellitus. In Liénaux's case

the pancreas was reduced to 3 cm. in length and 1 cm. in width, and in Sendrail's case it was shrunken to the size of a bean. In a case of atrophy of the pancreas in a dog observed by Müller there was emaciation in spite of the appetite being maintained; undigested muscle fibers were found in the feces and there was an absence of free hydrochloric acid in the stomach.

Mention must also be made of the following abnormalities that have been met with at postmortems. Calculi in the ducts of the gland cause dilatation of the ducts, an increase of connective tissue, and atrophy of the parenchyma. Calculi of the pancreas have been met with fairly frequently by Scheunert & Bergholz, especially in the cow. The presence of the following neoplasms has been recorded: adenoma (Liénaux), carcinoma of the head of the pancreas, especially in the dog, and melanoma in the horse (Bruckmüller, Friedberger, Käsewurm). Echinococci, larval sclerostomes and intestinal worms have occasionally been observed. Nencioni found chronic pancreatitis in an emaciated cat due to a colony of distoma felineum in the gland. In cattle in Cochin-China, Tonking, Java and Japan, Dicrocoelium pancreaticum occurs which causes a moderate induration of the walls of the glandular ducts.

Section VIII.

DISEASES OF THE PERITONEUM.

1. Ascites. Hydrops ascites.

Ascites is the term applied to the collection of serum-like fluid in the peritoneum not due to an inflammatory process.

Occurrence. Ascites occurs most commonly in the dog and generally as a result of disease of the heart or liver. The disease is seen in ruminants far more rarely. It is only quite exceptionally that the disease is observed in the horse, pig and birds.

The disease was found in ½% of 70,000 dogs examined by Fröhner. Cadiot saw 37 cases in three years at Alfort. Out of 28 cases 10 were due to disease of the heart and pericardium, 8 to tuberculosis, 4 to pleurisy, 2 to malignant growths in the liver and lungs, 3 to chronic interstitial hepatitis and one to cancer of the liver.

Etiology. *The principal cause of ascites unassociated with general dropsy of the rest of the body and of the subcutaneous tissue is congestion of the portal area.* This congestion may be due to compression of the portal vein by neoplasms, enlarged lymphatic glands or tumors of the pylorus or of the head of the pancreas. Under these and other conditions there is thrombosis of the portal vein. Portal congestion may also be set up by chronic diseases of the liver such as cirrhosis, echinococcosis, and neoplasms. In these cases the interlobular branches of the portal vein are subjected to pressure and the outflow of blood from the portal area is thus obstructed.

In cattle ascites is due most commonly to tuberculosis. Other chronic diseases of the peritoneum, such as neoplasms, may have a similar effect. These growths may cause obstruction of the mesenteric or portal veins or of the great lymph vessels and prevent the absorption of the lymph. The more common sequel to these lesions is chronic peritonitis.

As a part of general dropsy ascites occurs in cases of thrombosis or compression of the posterior vena cava between the liver and the heart, heart disease and chronic diseases of

the lungs. Although in these cases ascites is the result of passive congestion of the venous system throughout the body, or at least in the hind quarters, it very often remains the only symptom for a long time, especially in dogs.

Ascites and other symptoms of dropsy are often seen in animals that are cachectic. A similar form of the disease is seen in cases of chronic nephritis, liver rot, parasitic pneumonia and gastritis, or in cases where substances rich in water figure largely in the diet (turnips).

In young animals, and especially in young dogs, uncomplicated cases of ascites apparently occur. The fact that such cases often recover completely, suggests the uncomplicated nature of the condition.

According to Hamburger this is a specific condition caused by an organism named by him the "Bacterium lymphagogon" and which increases the quantity of lymph through the action of its metabolic products.

Anatomical Changes. In the horse there may be as much as 170 liters of liquid in the peritoneum (Brusasco) and in the dog twenty liters (Hördt). The liquid is sometimes clear as water and in other cases slightly turbid. It may be faintly yellow in color, watery, and contains at most only traces of fibrin. The specific gravity is below 1,016, but, as a rule, very nearly that; it contains 3.5 per cent of albumin and its chemical reaction is either alkaline or neutral. The fluid is often opalescent. By transmitted light it may have a yellowish tint and by reflected light a greenish color. This is probably due to the presence of blood pigment. When the transudation is due to a rise of pressure in the portal system the liquid may be from reddish to blood red in color. In cases of jaundice it is greenish and bile pigments can be demonstrated in it. There is, as a rule, only a small amount of sediment composed of a few white blood corpuscles, desquamated endothelial cells infiltrated with fat, granular débris, threads of fibrin, and not rarely a few red blood corpuscles. In some cases the latter are present in large numbers (Fig. 96).

The peritoneum appears thickened and shows adhesions at places where the disease has been in existence for a long time. The abdominal viscera appear anemic and even atrophied. In many cases some primary condition is discovered.

In cats and dogs the transudate is sometimes comparatively rich in fat (Ascites adiposus or chyliformis) and appears gray or milk white. The liquid may be translucent or quite opaque and when allowed to stand a thick layer of fat may form on the surface. The fat droplets are derived from desquamated endothelial cells that have undergone fatty degeneration or from the cells of neoplasms (cancer epithelioma), or directly from the blood in cases of lipemia. Wohlmuth found white colored serum in the peritoneal cavity of a fowl.

In many cases the fluid resembles lymph (ascites chylosus). Cases of this kind have been observed in cats by Dollar, Gray, Marcone, Wohlmuth and Suffran, and one case in the dog by Johne. The lymph may be derived from a ruptured

thoracic duct or from the lymph vessels of the intestinal wall as a result of peritoneal carcinoma. Nothing special could be found in the peritoneum in Wohlmuth's case. The fluid is milk-white, it contains a large number of lymph corpuscles, and on standing a creamy layer forms composed of fat droplets. Storch found that the liquid obtained from Wohlmuth's case was alkaline (in contradistinction to the amphoteric reaction of milk), the specific gravity 1015, the fat droplets were very minute and the percentage of fat was 7.6. There was a little sugar, fibrin, globulin and albumin.

Bennert, Joachim and others have shown that the milk-like appearance of transudates that occur in the peritoneal cavity of man is sometimes due to a pseudoglobulin which contains lecithin. In such cases the addition of ether and potassium hydrate does not cause the liquid to clear up.

Symptoms. The presence of large quantities of liquid in the peritoneum is evidenced by a distension of the abdomen. In emaciated animals this distension appears larger than it really is as it is in contrast with the wasted quarters. The more or less enlarged abdomen (Figs. 97, 98) varies in shape according to as it is looked at from before or behind. In the downward di-

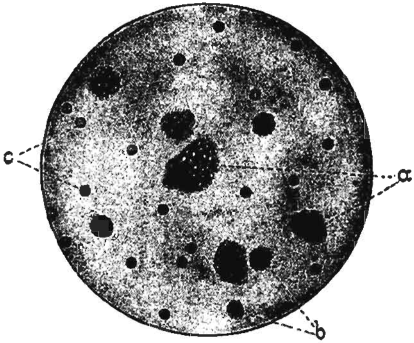


Fig. 96. Sediment of ascitic fluid. a. Endothelial cells infiltrated with fat, b. white corpuscles, c. red corpuscles.

rection it gradually increases and reaches its maximum in the neighborhood of the white line. The flanks fall in and the transverse processes of the lumbar vertebræ and the last ribs become prominent. In some cases the spines of the lumbar vertebræ form a convex line. In small short-coated animals the abdominal wall stands out behind the costal arch because this offers a greater resistance to the pressure of the liquid than the soft abdominal wall. The lower line of the abdomen becomes horizontal (Fig. 98) or curves downwards and extends below the line of the sternum (pendulous belly). In the dog the abdomen sometimes comes in contact with the ground and the umbilicus may appear as a hemispherical translucent projection. The

shape of the abdomen varies with the position of the animal. If a small animal be placed on its hind legs or in a sitting position the pelvic portion of the abdomen becomes prominent (Fig. 99). If the dorsal position be taken up, there is bulging of the flanks. Difficulty of respiration follows if the animal be held on its fore legs because the fluid presses on the diaphragm and hinders its movements.

Fig. 97. Distension of the abdomen owing to ascites.

On palpation the upper parts of the abdominal wall are found to be more or less tense, but the lower part is yielding. If the abdominal wall be not too tense intermittent pressure sets up undulating movements in the liquid which can be felt with the hand or even seen in some cases. In the larger animals the movements of the liquid can be appreciated by a hand introduced into the rectum.

Percussion reveals dulness extending upwards to various levels and the upper limit is sometimes marked by an undulating line. In small animals set up on their hind feet the dulness is found in front of the pelvis in the pubic region and if the animal be on its back, in the region of the loins or flanks.

In the larger animals auscultation over the lower part of the abdominal wall reveals no intestinal murmurs or very faint ones. The position of the area of dulness varies with the attitude taken up by the animal. By tapping the abdominal wall smartly with the flat of the hand or by shaking the animal's body, splashing sounds are sometimes produced which resemble those produced by the displacement of liquid gastric or intestinal contents containing large quantities of gas.

The extent to which the respiration is impeded depends upon the quantity of liquid. In order to take as much pressure as possible off the diaphragm and lungs, the animals, and especially dogs, prefer to take up a sitting position and to avoid all movement.

The compression of the stomach and intestines by the liquid causes constipation, tympanites and anorexia. In dogs the persistent pressure on the bladder sometimes causes the involuntary passage of urine (Spinola).

Other symptoms depend upon the nature of the primary disease, but, as a rule, there are, sooner or later, symptoms of anemia and debility, and finally complete exhaustion.

Course. Secondary ascites is a condition that lasts for a long time, whereas the apparently primary ascites of young dogs develops quickly and may disappear within a few weeks. Secondary ascites is variable, especially in the early stages. There may for a time be a complete disappearance of the liquid, but in the later stages there is a tendency for symptoms of dropsy in other parts of the body to make their appearance. In those cases in which the primary disease does not cause death the gradual increase in the quantity of liquid leads either to asphyxia or exhaustion.

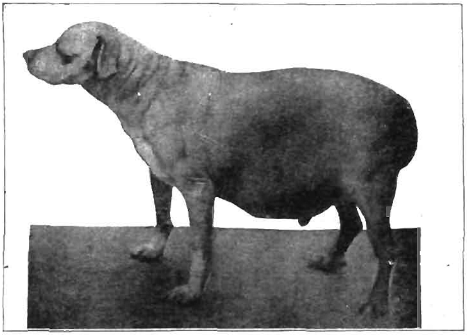


Fig. 98. Distension of the abdomen owing to ascites.

Diagnosis. Diagnosis is based principally upon the demonstration of the presence of liquid in the peritoneum. This, as a rule, presents no difficulties in the case of the smaller animals when the symptoms described above are taken into consideration. Similar alterations in shape of the abdomen may be produced by large cysts or cavities filled with liquid, such as the pregnant uterus, or uterus filled with fluid, dropsy of the fetal membranes, cystic ovaries or dilated bladders in small animals. Large soft tumors, such as myxomata, may also give rise to similar effects on palpation or percussion, and such growths may be capable of having their position altered by manipulation. In small animals careful palpation, especially in the neighborhood of the portal fissure of the liver, enables one

to feel the end of the cystlike body and, with very few exceptions, there is no change in the shape of the abdomen or in the position of the area of dulness in different positions of the animal. The loops of the intestines do not permit of any change of position of the large growth at all or only after some time. In the larger animals rectal examination furnishes valuable evidence for the exclusion of tumors or pregnancy. Retention of the urine is almost always observed in cases of distension of the bladder. Exploratory puncture is negative when there is a tumor. In cases of cystic ovary a viscid liquid containing ciliated epithelium is withdrawn and in hydro- or pyrometra the liquid is generally turbid or puslike and putrid. The differential diagnosis of obesity is easy, because alteration of the position of the animal does not affect the shape of the abdomen and extensive areas of dulness are lacking. Of course, the negative result of exploratory puncture requires an explanation.

Fig. 99. The distension of the pelvic and neighboring portion of the abdomen due to ascites, the animal being held up in a sitting position.

The presence of liquid in the peritoneum is not diagnostic of ascites, because the same condition is met with in serous peritonitis and as a result of rupture of the bladder. The differential diagnosis of acute peritonitis is easy on account of the pain on pressure on the abdominal wall, the elevation of temperature and the rapid course of the disease. It is more difficult to exclude the possibility of chronic peritonitis. The symptoms so closely resemble each other that a well-founded diagnosis can be based on the examination of the liquid obtained by puncture in cases where the primary disease and the manner of development of the condition are not known. Contrary to what is the case with the transudate (see page 502) the exudate is turbid, contains a large proportion of albumin (more than 3.5 per cent); its specific gravity is rather high (above 1,016) and it contains fibrin and is comparatively rich in white blood corpuscles. In cases of rupture of the bladder, the bladder is found to be empty and the expired air, the skin and the liquid obtained by puncture smell of urine.

It is often difficult to discover the immediate cause of the ascites. As a general rule simple ascites is due to some dis-

ease in the portal area, while dropsy of the other body cavities and of the subcutaneous connective tissue indicates stagnation of blood in the vena cava due to disease of the heart, lungs or kidneys, or it may indicate general hydremia. Exceptions to this rule are not uncommon. The pressure of the transudate on the blood- and lymph-vessels of the abdominal wall may cause subcutaneous edema. Diseases of the heart and lungs not rarely cause symptoms which for a time resemble those of ascites. A careful examination of all the organs allows some opinion to be formed as to the situation of the primary disease. The endocardial murmurs, irregularity of the heart and pulse are sufficiently diagnostic of diseases of the heart. A decrease in the quantity of urine passed is easily observed. The differential diagnosis of chronic diseases of the lungs is fraught with more difficulty since ascites also disturbs respiration.

Some diseases of the liver cause jaundice, while in other cases enlargement of the liver or a tumor in the portal space can be diagnosed by palpation. Removal of a part of the fluid greatly facilitates this examination in that the abdominal wall is relaxed and the contents of the abdomen are felt far more easily.

Prognosis. The prognosis is unfavorable, as a rule, because ascites is generally due to some incurable primary disease. Exceptions to this rule are cases of hydremia and cases of ascites due to excessive feeding with materials rich in water. In such cases a complete change of diet is sufficient to effect a cure, provided it be not too late. A more favorable prognosis can be given in cases of simple ascites in young dogs.

Treatment. Recovery promptly follows the removal of a part of the liquid in cases of ascites in young dogs. Exceptionally similar recoveries take place in the horse and ox (Brusasco, Hajnal, Fekete). The ascites occurring in socalled dropsy of the cellular tissues (Zellgewebswassersucht) of cattle is cured by the administration of dry food, provided the case is not too far advanced. Good hay, alfalfa and corn should be given.

In practically all other cases treatment is symptomatic and aims at relieving the functional disturbances caused by the collection of the liquid. It is, as a rule, only dogs and other pets that offer the opportunity for treatment, this being rarely undertaken in animals that can be slaughtered or in horses.

The absorption of the transudate is assisted by diuretics; infusion and other preparations of digitalis (see page 666), strophanthin (see page 666), sodium benzoate or citrate of caffeine are valuable The salts of caffeine may be given (to dogs) in doses of 0.1-2 grams three or four times a day. The following diuretics should also be mentioned; potassium acetate in doses of 0.1-0.2 grams three or four time a day, the infusion of digitalis, juniper juice (1.0-2.0 gm.), squills either as

powder or infusion, diuretin (0.5-1.0 gm. every two hours), agurin (1.0-2.0 gm. daily), theocin (0.6-1.2 gm. per fifty kilos of body weight [Albrecht]). Purgatives are less to be recommended on account of their less certain action and the debility of the animal. Besides the neutral salts, calomel is the drug principally employed. Small doses (0.3-0.4 gm. for dogs) are given, and if necessary combined with jalapin.

Provided that the condition of the heart and lungs permit of its use, injections of pilocarpine produce more favorable results by stimulating diaphoresis and the secretion of large quantities of saliva. The dose that may be employed for dogs is 0.005-0.1 gm. in watery solution and this may be repeated daily. According to Zahns five to ten drops of a 1 per cent solution may be given three times a day. Arecoline may be used in some cases.

Puncture is indicated in cases where the quantity of liquid is considerable and this may have to be repeated on account of the appearance of symptoms of dyspnea. Even repeated tapping is borne moderately well if it be possible to compensate for the loss of albumin by suitable nourishment.

Tapping is practiced at the lowest part of the abdomen with the animal in the standing or if necessary in the prone position. After proper preparation of the seat of operation (shaving and washing of the skin with alcohol or ether) the abdominal wall is punctured with a sterilized trochar of not too wide a bore, or with the needle of a hypodermic syringe. The stiletto is withdrawn and the tap of the canula closed. To the canula is attached a long piece of rubber tubing, the free end of which is put into a clean glass vessel placed on the ground. The cock is then opened and the liquid allowed to run out. If the flow ceases movements are made with the canula in order to displace the piece of mesentery or loop of intestine that is causing the obstruction. Should this prove unsuccessful the rubber tube is removed and a sterile probe is introduced in order to remove any pieces of fibrin that may be sticking in the canula or to displace any organ that may be covering the opening of the canula. When the liquid has been withdrawn the wound in the abdominal wall, which is as a rule covered by the skin which was displaced before the deeper layers were penetrated by the trochar, is sealed either with collodion or with a pledget of wool soaked in pitch. It appears to be advisable to put on a pressure bandage. Dieulafoy's suction pump may be used for the withdrawal of the liquid.

Tapping needs to be carried out with care and as slowly as possible as a too rapid removal of the liquid may lead to rupture of blood vessels, dangerous disturbances of function of the heart or acute cerebral anemia. Should there be a sudden appearance of symptoms of collapse during tapping this must be stopped immediately and injections of ether or camphor given, and if necessary black coffee may be administered. At each subsequent tapping a fresh seat of operation must be selected.

To prevent fresh effusion of liquid, French authors (Pressecq, Sansot) advise intraperitoneal injections of tincture of iodine or Lugol's solution. This treatment ought to give the best results in a primary case, but it is dangerous in that it may cause a rapidly fatal peritonitis. More recently the intraperitoneal injection of a 1 in 5 solution of choral hydrate solution has been advised by Sendrail & Cuillé. Directly a portion of the transudate has been removed from the abdomen a syringe is connected to the rubber tube and the sterilized watery solution is introduced (about 1 gm. per 3 kilograms body weight). As a rule several injections are necessary at increasing intervals. The authors record good results with this treatment.

Omentopexia (Talma's operation in human surgery) is also recommended by Sendrail & Cuillé. This involves the insertion of a piece of the great omentum measuring about a hand's breadth between the oblique muscles of the abdominal wall, thus establishing a collateral outflow from the portal system into the general circulation. Satisfactory results were obtained in three cases treated in this way.

2. Peritonitis.

Occurrence. Peritonitis is, as a rule, a secondary condition and occurs most frequently in the horse, owing to the susceptibility of this species to severe colic of different types. Next to the horse, the ox, and especially the cow, is likely to suffer from peritonitis, puerperal infection and traumatic gastritis being the primary diseased conditions. The disease is far more rare in the other domestic animals and in birds.

Etiology. Apart from some cases that are chronic from the outset, the real cause of peritonitis is an infection of some sort. Several varieties of bacteria are capable of setting up the disease. The bacillus bipolaris in cases of hemorrhagic septicemia (see Vol. I) is a well-known cause of peritonitis. In these cases the inflammation is generally slight. The pyogenic bacteria, viz. staphylococci, diplococci and streptococci and the bacillus coli communis which is always present in the intestine, are frequent causes of peritonitis, the latter especially in cases that are associated with disease of the intestine (Borszéky & Genersich, Hensen). The tubercle bacillus, bacillus pyogenes, streptothrix (actinomyces) canis, in some cases the anthrax bacillus, the bacillus of swine erysipelas (Eisenmann) and other organisms may give rise to peritonitis. In fowl cholera the peritonitis is sometimes so prominent a lesion that it may be considered as an independent condition.

Preisz records a case of peritonitis and pericarditis with local necrosis at the seat of inoculation in a horse vaccinated against anthrax. A rounded or short oval bacillus was cultivated from the blood which set up acute inflammation of the serous membranes after subcutaneous inoculation into experimental animals. The bacteria reached the peritoneum from the seat of inoculation by way of the blood stream.

In the exudate from a case of serous peritonitis in a horse Hamburger found streptococci (Str. peritonitidis equi). Intraperitoneal inoculation of cultures into a horse set up serous peritonitis accompanied by fever.

Injuries to the abdominal wall are very frequent causes of peritonitis (peritonitis traumatica et operativa) if the peritoneum be injured, because the peritoneal cavity is laid open to the entrance of infective material. Violent blows with blunt objects are likely to set up at most a circumscribed peritonitis only. Besides accidental wounds of the abdominal wall, those made in the course of operations play an important part in the production of peritonitis. Among these may be mentioned laparotomy, operation for hernia, castration, etc. Simple puncture carried out with all precautions as to asepsis is far less dangerous. Horses are particularly susceptible to infection in this way and this fact is no doubt associated closely with the high degree of susceptibility that horses, as a rule, show to the pyogenic bacteria.

Microorganisms which cause inflammation reach the peritoneum from abdominal organs which contain them, most easily if a break in continuity occurs in these organs (Peritonitis

perforativa). Especially in cattle, foreign bodies often lead to such a peritonitis. Other causes are tearing of the rectum during rectal examination, during the administration of enemata, or during coitus; rupture of vagina or uterus during difficult labor and particularly during instrumental interference; rupture of the oviduct in birds; rupture of the urinary bladder in retention of the urine or if it is perforated by the catheter; rupture of the gall bladder, etc. Similarly peritonitis develops after rupture of stomach or intestine as also after rupture of other abdominal organs if these contain bacteria owing to a preceding disease, for instance in suppuration of the liver, spleen, prostate, testicles or spermatic cords, in the mesenteric lymph nodes, in the uterus (puerperal metritis).

In bacterial inflammations of the abdominal organs the inflammatory process may extend to the peritoneum without there being any rupture of a viscus, the infection reaching the peritoneum either directly or by way of the lymph stream. In this connection deep-seated inflammation of the stomach, intestine and interior of the bladder or sexual organs are most dangerous.

Bongartz frequently observed peritonitis in cows newly brought into cow houses and thought that it was connected with active fermentation processes in the intestine, the products of the fermentation favoring the passage of the bacteria through the intestinal wall.

Pleurisy very rarely sets up peritonitis by extension.

In the group of acute infective diseases known as the hemorrhagic septicemias, peritonitis is comparatively common, but in the specific septicemic diseases, such as anthrax, swine erysipelas, etc., diffuse peritonitis is rarely met with.

Finally, peritonitis may be caused by bacteria carried by the blood stream from lesions in peripheral parts of the body.

Peritonitis associated with gastritis or septicemia has often been observed in sheep and cattle that have been fed on bad turnips.

In Upper Italy, Boccalari saw cases of rapidly fatal sero-fibrinous peritonitis in calves from 2 to 4 months old. Organisms resembling Fränkel's diplococcus were found in the exudate. The organism was fatal to mice and rabbits in 24 hours. Material taken from experimental animals was non-pathogenic for calves but the exudate from diseased calves was proved to be pathogenic.

In birds hyphomycetes (Aspergillus fumigatus) is sometimes responsible for peritonitis. Lignières & Petit observed an epizootic peritonitis of this nature in turkeys. Gougerot & Caraven saw two cases of peritonitis in young dogs due to the Sporotrichum Beurmanni.

Chemical substances, such as bacterial toxins and materials retained in the blood as a result of nephritis, are very rarely responsible in themselves for peritonitis. Sterile bile, urine and other liquids do not cause the disease. Occasionally animal parasites are responsible for peritonitis, this having been observed in carnivora by Baillet, Railliet, Labat and Cadéac.

Very occasionally cases have occurred in cattle and pigs as a result of a severe invasion of Cysticercus tenuicollis or the liver fluke.

Chronic peritonitis is generally due to chronic or repeated attacks of sub-acute inflammation in neighboring organs, whereas external or internal injuries, as a rule, set up a localized peritonitis which results in adhesions of the peritoneal surfaces. According to the observations of Oppermann, there often develops in draft oxen a peculiar form of chronic peritonitis with numerous and extensive adhesions of the stomach and intestinal loops, abscess formation, and the collection in the abdominal cavity of large quantities of a foul-smelling, turbid, discolored exudate containing flocculi of fibrin. This condition results from repeated chilling of the abdominal walls when, during the cold and wet days of autumn, the animals are fed in the field and after feeding lie down on the cold, wet ground. On the other hand, Oppermann could not substantiate the assumption of Fuchs, according to which the inflammation in such cases is caused by feeding ensilaged turnip chips or large quantities of chaff, since cows and other animals fed on such feed do not sicken.

Chronic peritonitis is seen in tuberculosis, glanders and accompanying carcinoma, sarcoma and other neoplasms of the serous membrane. It may also occur in animals that are in a debilitated condition and as a result of ascites. The frequently occurring cases in horses associated with the formation of false membranes are due to an antecedent invasion by larval sclerostomes (Glage).

In the causation of acute primary peritonitis, which occurs without an injury to the peritoneum and without a primary affection, and sometimes in certain secondary cases of peritonitis, predisposing affections may be concerned, such as taking cold (P. rheumatica s. frigore), weakening influences, blunt traumatic injuries to the abdominal walls, etc.

Pathogenesis. Although the ability of the peritoneum to dissolve bacteria is considerable, the localization and multiplication of microorganisms which reach the peritoneum becomes possible partly owing to an accidentally great virulence or massiveness of the infection, partly because the peritoneum is injured by bacterial toxins of unusual toxicity or by products of inflammation or cleavage which have reached it from other organs; finally owing to considerable injuries of the peritoneum and to hemorrhages in protracted operations and from trauma. In these cases the microorganisms in part escape bacteriolysis and absorption, they multiply actively and give rise to inflammation. Through the irritation produced by the toxic bacillary products, the blood vessels of the peritoneum are dilated, a liquid exudate mixed with leucocytes (so-called early exudate) forms and is soon followed by coagulation, usually leading to

adhesions between the several abdominal organs. The exudate is at first slight and may disappear entirely, but the formation of a fibrinous exudate continues, especially in circumscribed peritonitis. According to the virulence and the number of the bacteria, and according to the bacteriolytic activity of the peritoneum, a general or a circumscribed peritonitis develops. An inflammation which at first is circumscribed may, however, become general later on.

The inflammatory irritation of the nerve endings in the peritoneum, which has a large nerve supply, is responsible for the uninterrupted pain of inflammation which is present in acute peritonitis; it is increased by pressure and also if the abdominal organs change their position. As the peritoneum possesses a great power of absorption, the microorganisms and their endotoxins, and in case of perforation-peritonitis also other toxic substances are transported into the general circulation in considerable amounts, and in this manner general disturbances are caused which correspond in intensity to the extent of the inflammatory process. Peritoneal intoxications of this kind may lead to death in cases with extensive perforation of abdominal organs, even before the peritonitis is fully developed. At first the intestinal movements are stimulated by the inflammatory irritation, but later they are impeded by reflex action of the sympathetic nerves; occasionally they may be inhibited by serous infiltration of the intestinal muscles, if the peritonitis is severe. Accidental elimination of certain bacteria and of their toxins through the intestinal mucosa, or disintegration of the intestinal contents may give rise to a different behavior on the part of the peristaltic motion.

Anatomical Changes. In the early stages the peritoneum is markedly congested, dull and opaque, and is frequently studded with punctiform hemorrhages. Within the next twenty-four hours there form thin filmy fibrinous membranes which are easily detached. These may subsequently measure several millimeters in thickness, and while still remaining soft and easily torn, cement opposed layers of the peritoneum together. There is a simultaneous accumulation in the peritoneum of a turbid serous fluid containing flocculi of fibrin. In the horse there may be as much as forty liters of such exudate and in cattle the amount may be as much as 100 liters. In other cases the exudate is purulent. In cases of rupture of the stomach or intestine the exudate is mixed with food or intestinal contents and in cases where the perforation is effected slowly the exudate is sanious. In any case the exudate may contain a larger or smaller proportion of blood. Chronic peritonitis is characterized by the formation of connective tissue, whereupon adhesions are formed between the different abdominal organs or between these and the abdominal wall.

The encapsuled abscesses in cattle and pigs due to infection with the bacillus pyogenes are produced in this way. Apart from this, acute fibrinous peritonitis usually becomes chronic with the development of connective tissue under the fibrinous membranes. The peritoneum becomes thickened and the surface of the organs is covered with thick layers of connective tissue. Both these forms are seen very often in the liver and spleen of the horse. Chronic peritonitis is very often circumscribed in extent, especially on the serous membranes of the liver and spleen, seats of puncture of the rumen or cecum, and in the pelvis around the bladder and sexual organs. In cattle, and still more frequently in pigs, chronic peritonitis leads to the formation of bony plates close to the wound made for ovariotomy (Gurlt, Johne, Delaud).

Symptoms. In view of the fact that peritonitis is generally a secondary condition, its symptoms are preceded by those of the primary disease, but the latter give way to those of peritonitis either gradually or suddenly, according to whether the infection of the peritoneum is severe or not. The primary disease is, as a rule, *some severe disease of the stomach, intestine* or genital organs. If the peritonitis is due to an acute septicemic disease its symptoms are more or less obscured by those of the primary disease, but in other cases the symptoms of peritonitis are very obvious.

In cases of **acute diffuse peritonitis** abdominal pain is the most pronounced and usually the most persistent symptom. Animals show evidence of pain in a variety of ways, groaning, whining, bellowing, grinding the teeth, looking round at the body, *switching their tails, etc.* They avoid all sudden movements, stand with their feet close under the body, either arch or hollow their backs, and in the latter case hold their heads and necks drawn back. Horses often try to lie down, but, as a rule, do not get beyond the attempt; should they succeed, however, they exercise great care in carrying out the movement, and in some cases lie on their backs. Cattle, as a rule, remain standing and only lie down just before death. Small animals lie in a crouching attitude, and their countenances are often expressive of great pain. The abdominal wall may be painful all over, or only in parts, and the animals try to avoid all pressure on it. Motion is stiff and only short steps are taken, any deviation from the straight line being avoided. If the inflammation is localized in the anterior part of the abdomen, going uphill is especially painful.

The abdominal pain and the susceptibility to pressure of the abdominal wall vary from individual to individual, but to a still more marked degree in the various species. In the horse the symptoms of pain are principally subjective, and they are never completely absent; whereas susceptibility to pressure on the ab-

dominal wall is very seldom demonstrable, owing to its natural and in this case increased tenseness. In small animals the opposite holds good and in cattle both subjective and objective symptoms are less clearly shown and may be absent altogether.

The abdomen may be less than normal in circumference in the early stages owing to reflex contraction of the muscles. Later in the disease the accumulation of a copious exudate gives rise to enlargement of the abdominal circumference and causes horizontally limited dullness.

Friction sounds can be heard only rarely. They are most frequently observed in cattle and in the left hypochondriac region if the inflammation involves the diaphragm (foreign body), and in the right flank in cases of metro-peritonitis (Detroye). The roughness and painfulness of the peritoneum can easily be discovered in larger animals by rectal examination, and in many cases of rupture of the stomach or intestine, particles of food can be found in the peritoneum.

Peritoneal adhesions may be recognized by the limited mobility of the rectum or the nonmobility of the nonpregnant uterus, while in cattle the inpalpability of the contents of the rumen may indicate a simultaneous presence of a gelatinous thickening of the great omentum (Zieger).

Neighboring organs are frequently affected in sympathy. In carnivora and swine there is often, exceptionally in horses, vomiting. On the other hand, extension of the inflammation to the diaphragmatic layer is sometimes followed by persistent hiccough (Singultus). In cattle Otto noticed peculiar rolling sounds following every movement of the rumen.

In the early stages of the disease there is diarrhea, and active intestinal murmurs can be heard, provided the primary disease is not one in which peristalsis is suppressed. Very soon and in some cases from the outset, constipation sets in which is of a very persistent character. In the later stages there is general meteorism. Frequent and painful straining to defecate is one of the common symptoms of general acute peritonitis. Usually micturition is frequent with pressure of urine and tenesmus and forms an important diagnostic sign particularly in peritonitis with perforation in horses. Exceptionally only is retention of urine observed in this affection.

The respiration is costal and accelerated. With the increase of the exudate and the meteorism the difficulty of respiration becomes more pronounced.

In acute peritonitis in cattle Cuny observed a bilateral flow of tears which soon became converted into a purulent discharge, associated with redness and swelling of the conjunctiva and opacity of the cornea. He also noticed a watery nasal discharge which in the later stages became mucoid.

Among the symptoms of general disturbance, which are al-

ways present, fever is very rarely absent and is, as a rule, high. The fever takes no typical course (Fig. 100). In carnivora and more rarely in other animals the temperature at the onset and even up to the end may be subnormal in cases of perforative peritonitis. The pulse, which is quite independent of the temperature, is without exception accelerated from the outset and may be twice as rapid as normal or even faster. In the early stages it may be rather tense, but soon becomes very weak and may be nearly if not quite imperceptible, probably on account of the serious fall in blood pressure. To this are due the sudden cooling of the extremities (especially the ischial region and root of the tail [Otto]), the cyanosis and also, at least to some extent, the languor and prostration. There is, as a rule, a complete absence of appetite.

In severe septic conditions (septic metritis, rupture of an abscess) peritonitis develops rapidly with the symptoms of pyemia or septicemia, there being high fever and languor with the early onset of diarrhea, but no symptoms of pain.

In cases of rupture of the stomach or intestine there is a sudden depression, the extremities are very cold, the pulse is thready and very rapid and the body is bathed in cold sweat. In such cases the excessive tenseness of the abdominal wall, the suddenly developing distension followed by a rise of temperature, the rough condition of the peritoneum which is sometimes discoverable by rectal examination, and the complete collapse indicate peritonitis.

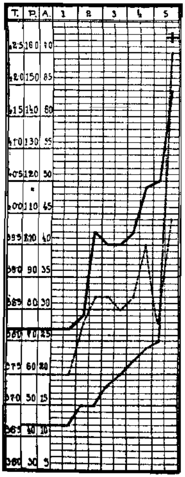


Fig. 100. Temperature chart of a case of secondary diffuse peritonitis in a horse due to injury of the interior of the intestine by a calculus.

In cases of **circumscribed acute peritonitis** the pain is generally less severe and localized. *The general symptoms* are less pronounced and the principal symptoms observed are due to disease in neighboring organs. Such localized peritonitis may lead to the formation of adhesions and the encapsulation of the exudate, but there is always the danger that under certain circumstances there may be a general peritonitis spread from such places.

The symptoms of **chronic peritonitis** are, as a rule, slight and indefinite; gradual wasting associated with variable temperature, digestive disorders with persistent diarrhea, while in the horse occasional transitory attacks of colic. Diagnosis is generally possible only if in addition to the above mentioned symptoms there is localized sensibility to

pressure of the abdominal wall, if there is an accumulation of fluid, which proves to be an exudate on test puncture.

According to Oppermann, chronic peritonitis in oxen takes a slow course, resulting in disturbance of appetite, slight fever, light symptoms of colic, and diarrhea lasting from one to two days. After several such light attacks the animals will lose condition in spite of an improved appetite; the hair becomes dull and rough; they continue to strain for a while after defecation, air being sucked in through the relaxed anus and expelled again with a blowing sound (so-called blowers). During such periods, which last for months, the animals become greatly emaciated, while the circumference of the abdomen becomes greatly increased, particularly in the subchondrial region. Exacerbations continue to recur under the influence of overexertion or the consumption of large quantities of feed. Further symptoms are groaning while stepping around or while executing movements, or when the animal stands for unusually long periods of time, sensitiveness to pressure of the abdominal walls, and sunken eyeballs. Upon rectal examination, the free mobility of the rectum is absent. In the afebrile course an aggravation of the condition finally takes place, accompanied by a drop in body temperature and slight bloating, death following in from 7 to 8 days.

Peritonitis following a parasitic inflammation of the oviduct in chickens is characterized by restlessness, droopiness, and the laying of thin-shelled and finally soft-shelled eggs. Laying is soon discontinued and death follows in a few days.

Localized chronic peritonitis frequently causes disturbance of other organs (loss of tone of the rumen in cattle, stenosis of the intestine). The exact nature of these derangements can be determined in rare cases only by rectal examination or palpation of the abdomen.

Course. Acute peritonitis is sometimes very rapid in its development and may cause death in the first stage of the disease and even within a few hours. This is particularly the case after perforation of abdominal organs, and exceptionally in cases of puerperal infection. In such cases the local symptoms are less pronounced and the symptoms shown are those of general sepsis. In the great majority of cases the symptoms develop slowly, death taking place from the fourth to fourteenth day. Sometimes there is a marked fall in temperature before death and there is usually diarrhea. In other cases the disease becomes chronic and may last for months or even years. The same is true for cases that are chronic from the outset.

Diagnosis. The moderately rapid development of the disease, fever, tenderness of the abdomen to pressure, constipation and tympanites, acceleration of the pulse from the commencement and gradual weakening of the heart beat form a

fairly characteristic picture. Diagnosis is more certain in cases where a primary disease is known to be in existence. Otherwise it can only be based on the discovery of a roughened condition of the peritoneum, adhesions between various organs or the presence of a fluid exudate in the peritoneal cavity.

In the horse the disease is likely to be confounded with gastric and intestinal colic in which the pulse is rapid and weak from the outset, or the animals are inclined to be persistently restless. In gastro-enteritis there is diarrhea either throughout or at any rate for a considerable time and loud intestinal murmurs, but rectal examination reveals no roughness of the peritoneum nor is it painful. Other kinds of colic can be differentiated by the facts that in them there is no rise of temperature for some time at least, and that the animals are restless. and rectal examination affords in many cases much valuable information. All forms of colic may lead to peritonitis in the later stages, and especially those due to thrombosis and to displacement of organs. In the dog cases of volvulus of the stomach may be confounded with peritonitis (see page 160). As a rule chronic peritonitis can only be diagnosed when the presence of fluid can be recognized in the peritoneum, or there is known to have been an antecedent acute attack. At most, limited areas of inflammation of the peritoneum will be found where there have been some adhesions formed (see stenosis of the intestine). The differential diagnosis of chronic serous peritonitis and ascites has already been dealt with.

Prognosis. Acute diffuse peritonitis must be considered as a very dangerous condition and the prognosis is very unfavorable when the inflammation is due to perforation of the stomach or intestine, or to the escape of pus into the peritoneum. Less extensive localized fibrinous inflammation is commonly unassociated with any danger, and is in many cases not recognized during life. Prognosis is more favorable in cases due to injury from the outside, because with suitable treatment the disease may be kept localized and a cure effected. Extensive chronic inflammation leads to adhesions between the abdominal wall and some of the viscera, especially intestine, and consequently digestive disorders of a permanent nature are produced.

Treatment. In cases where the peritonitis is due to some wound, such as castration, antiseptic treatment immediately after the first symptoms make their appearance will frequently arrest the extension of the disease and limit it to the immediate neighborhood of the wound. Surgical intervention may be followed by good results in cases due to encapsuled abscesses, such as are produced in cattle by foreign bodies. In certain cases of secondary peritonitis, cures might be effected by surgical intervention, such as laparotomy, suturing of the wound in the intestine, resection of the intestine, washing out the peritoneum

with sterile salt solution at body temperature, or with weak solutions of salicylic or boric acid. Baldoni cured a dog in seven days by flushing the peritoneum.

To decrease the absorptive power of the peritoneum Wilms advises in human surgery the intraperitoneal injection of 130 cc. of oil of champhor. Keller obtained the same result by applying 50 gm. of sterilized paraffine oil, evenly all over the peritoneum, by means of pledgets of cotton, if necessary, after first flushing the abdominal cavity with physiological salt solution. In four female dogs treated in this manner for purulent puerperal peritonitis (two of them associated with rupture of the uterus) three were saved. The absorption is impeded also by adrenalin, and by systematic applications of cold compresses which may moreover reduce the inflammation; similar effects follow inunctions of gray ointment on the inner surface of the thighs and on the abdominal wall (for horses, 5.0 gm.; dogs, 1.0 gm. daily as long as no salivation occurs). Not until the pain and the other symptoms diminish, are Preissnitz' applications or inunctions with oil of turpentine to be applied.

Attention should next be paid to decreasing the movements of the intestine to avoid as far as possible the dissemination of the infective material by the peristaltic action. This object is best attained by the administration of opium in doses of 5-10 gm. for horses and cattle, 1-3 gm. for small herbivora and 0.1-0.3 for dogs. Morphine hydrochloride may also be given in doses of 0.3-0.4 gm. or 0.02-0.1 gm. subcutaneously once or twice daily. Provided the primary disease permits of the administration of food this should be easily digested and if possible in fluid form (gruel, soup, milk). Mild purgatives, such as castor oil (horses 250-500 gm. and dogs 15-30 gm.) and calomel (dogs 0.03-0.05 gm.) are indicated for the constipation during the early stages. Lukewarm clysters are advantageous. Cattle should be given, at intervals of two to three hours, water, barley water, oatmeal or linseed tea to prevent the drying up of the food.

To increase the blood pressure one may use caffein, camphor, adrenalin, suprarenin, paranephrin, tonogen, and, according to Thun, in cows, inflation of the udder with air.

Tapping is indicated for the removal of the fluid and this may be repeated as often as necessary. In some cases puncture of the stomach or intestine may be necessary. In chronic peritonitis attempts may be made to promote the absorption of the exudate by Priessnitz' poultices and massage, potassium iodide (5-10 gm. for large animals or 0.3-0.5 gm. for small animals daily) and diuretics. Any derangements in other organs must be treated.

3. Tumors of the Peritoneum.

Occurrence. In addition to tuberculosis and actinomycosis true neoplasms may occur in the peritoneum primarily and

metastatically. Diffuse carcinoma and more rarely sarcoma (melanotic) of the peritoneum are seen sometimes. The membrane is beset with neoplasms of various sizes and the connective tissue layer appears to be thickened, while the neighboring viscera may be more or less compressed or atrophied. Fibromata, lipomata and myxomata, and very exceptionally angiomata occur in the peritoneum. They are practically always attached either to the omentum or mesentery and are, as a rule, solitary. The emphysematous cysts of the mesentery frequently found in the pig and due according to Jäger (A. f. Tk., 1906, XXXII, 410) to a bacillus of the colon group, the bacillus coli lymphaticum ærogenes, are of no importance from a clinical point of view. The same condition was found by Günther in a fowl.

Symptoms. As a rule neoplasms cause only disturbances of a general nature and possibly also abdominal pain, especially carcinoma and sarcoma. There is gradual wasting until a condition of extreme debility is produced. In certain cases the symptoms present indicate stenosis or occlusion of the intestine, the cause of which can only be determined by the positive result of palpation or rectal examination. The symptoms of chronic peritonitis or ascites are more valuable in this respect. Chylous ascites (see page 503) in particular raises the suspicion of carcinoma of the peritoneum. This suspicion is strengthened if a primary growth can be discovered in any other organ, such as the testicle, udder or prostate.

Treatment. In large animals and in cases where the neoplasm is situated in a part of the peritoneum accessible to operation, surgical intervention is the only treatment possible. No good results are to be expected if the growth be of a malignant nature.

4. Animal Parasites Found in the Peritoneum.

The Filaria papillosa occurs very frequently in the peritoneal cavity in the **horse** but it is quite harmless. Occasionally echinococci are found. In a case recorded by Toutey a cyst hanging by a pedicle was the cause of a twist. Cysticercus fistularis (Rudolph, Reckleben) which, according to Neveu-Lemaire, is probably identical with C. tenuicollis, and the larvæ of sclerostomes also occur. The latter may be either encapsulated in connective tissue or in hemorrhagic foci in the subperitoneal tissue, or there may be fully developed specimens free (Kitt).

In **ruminants** the Cysticercus tenuicollis, (see page 347) occurs with great frequency and may cause acute peritonitis if the infestation be excessive. The liver-fluke (Morot) and the Filaria labiopapillosa (Alessandrini) occur very rarely.

In the **pig** echinococci, Cysticercus tenuicollis and (in America) the thread-like Stephanurus dentatus are found.

In **carnivora** the following parasites may be found, echinococci (Railliet, Reinmann and Pécard, who in one case, found several thousand, some free and some attached to the peritoneum), Plerocercoides

Bailleti (found by Baillet and Cadéac each in a cat and by Labat and Railliet in dogs, sometimes in association with ascites, and in one case the liquid obtained by tapping contained large numbers of cysts), and finally the Pentastomum denticulatum (found by Roche-Fontaine in large numbers enclosed in cysts).

Eustrongylus gigas was found, usually in the abdominal cavity, in 0.37% of the dogs examined in America. Usually only a single specimen 35 to 102 cm. long (female) and 25 to 30 cm. long (male) was found. The animals almost never showed symptoms of disease, in spite of the presence in some places of peculiar changes of the peritoneum, such as chronic peritonitis with a greenish or dirty brownish, odorless, firmly attached fibrinous exudate consisting of polynuclear and mononuclear leukocytes. Furthermore, adhesions between the great omentum and the liver, spleen, pancreas, and intestines can frequently be observed.

In the Revue Vétérinaire (1906, 141), there was described by Sendrail & Cuillé under the term "Hydrops ascites parasitarius," a disease of the dog caused by worms of unknown origin. The disease occurs occasionally in the neighborhood of Toulouse, one or two cases coming under observation in the clinic every year. The disease differs from ascites clinically only in that the serous liquid in the peritoneum contains numerous cyst-like bodies which vary in size from a pin's head to a pea, are of variable shape and easily crushed, and which on standing separate out into a pasty mass from the otherwise clear liquid. They are motile. The disease may be transmitted experimentally by the introduction of living cysts into the peritoneal cavity of dogs.

According to Sendrail & Cuillé it is an abnormal form of the cyst of the Mesocestoides lineatus which is frequently found in the intestine of dogs in the neighborhood of Toulouse. The numerous cases observed in the past by Neumann and classed as echinococcosis should possibly have been described as this disease. Neveu-Lemaire mentions this parasite under the name of Dithyridium elongatum (Cysticercus elongatus) as the evolutionary form of a taenia which was unknown hitherto.

Diseases of the Respiratory Organs

SECTION 1.

DISEASES OF THE NASAL CAVITIES AND OF THE ACCESSORY CAVITIES OF THE NOSE.

I. Hemorrhage from the Nose. Epistaxis.

(*Haemorrhagia narium, Rhinorrhagia.*)

By hemorrhage from the nose proper is understood a hemorrhage from the vessels of the nose or from those of the accessory nasal cavities.

Etiology. Epistaxis is sometimes seen in otherwise perfectly healthy horses in consequence of a congenital predisposition, probably hemophilia, and may then occur without any obvious cause at all or from a quite insignificant external cause. Predisposition of this kind to epistaxis appears to be congenital, particularly in highly bred horses, and it often disappears with advancing age.

The most common causes of epistaxis are traumatic injuries to the head, such as a blow or a thrust, or injuries to the nasal mucosa, which may also be due to the introduction of the rhinolaryngoscope, to convulsive expiration after the penetration of foreign bodies, or to dust from the feed (Bigoteau), to stings and bites of parasites (Pentastomum, Œstrus), to laceration of the vena pterygoidea, or of the vena maxillaris interna in fracture of the condyloid process of the maxilla. Epistaxis also frequently occurs in fracture of the base of the skull. Another cause may be congestion of the head or stasis, especially in chronic diseases of the heart and lungs, also compression of the jugular vein.

Rare causes of epistaxis are teleangiectasia or angioma; the erosion of individual vessels in ulceration of the nasal mucosa commonly leads to hemorrhage from the nose, particularly also in glanderons ulcers. Rare canses of epistaxis are: laceration of the arteria pharyngea (Blaise), of the dilated arteria carotis interna, of the arteria maxillaris interna (Walley, Cadéac) or of the arteria nasalis (Marafon).

Epistaxis finally occurs not infrequently in the course of general acute infectious diseases (anthrax, purpura hæmorrhagica, scorbutus, smallpox); also in blood diseases (leukemia, anemia) and in dochmiasis in dogs.

Symptoms. There is a discharge of blood from one or both nares, either in the form of drops (epistaxis) or in a continuous thin stream or in larger amounts (rhinorrhagia); the blood is usually bright red, not foamy, perhaps mixed with a few large air bubbles. If nasal catarrh exists simultaneously, the blood may appear in the form of fine streaks in a mucoid or purulent discharge. If there is a more severe loss of blood, the patient is frightened, the pulse is small and frequent, the mucosæ are very pale and a fatal issue due to loss of blood may occur under intensification of the symptoms within eight to twelve hours, or only after five to eight days. In the majority of cases, however, hemorrhage ceases spontaneously after a time. We generally need consider only such hemorrhages as dangerous which occur frequently in anemic animals or which are due to serious underlying infections.

Diagnosis. It is usually easy to recognize the source of a hemorrhage in the nasal cavities or the accessory naval cavities. However, the cause of such hemorrhage can only be determined after a consideration of all concomitant circumstances and after a careful inspection of the nasal cavities, with the rhinolaryngoscope when possible. If there is a more severe hemorrhage such inspection must, however, be postponed, and the stopping of the flow of blood is the first requirement. In hemorrhage from the lungs the blood is likewise bright red; but it is finely foamy and there is cough and frequently dyspnea. Exclusion of hemorrhage from the lungs may be difficult in those cases of profuse bleeding in which portions of blood get into the pharynx and from there into the trachea or bronchi, causing cough and becoming more or less foamy. In hemorrhage from the stomach the blood is dirty brown, has an acid reaction and may in horses, and occasionally in cattle, be expelled from the nose with vomitory movements. Blood oozing out of ulcers is mixed with mucoid, purulent or ichorous discharges, if severe bleeding does not occur.

Treatment. Hemorrhage frequently stops soon and spontaneously if the animal is taken to a quiet place. If it persists, cold applications to the frontal region and to the nose are indicated, also astringent irrigations of the bleeding nasal cavity (alum, tannic acid, iron sulphate, chloride of iron in 3-5% solutions). In severe hemorrhage, which cannot be controlled otherwise, one or both nasal cavities must be tamponed; this may be done with absorbent cotton soaked in an 8-10% solution of chloride of iron, or with oil of turpentine or with a 0.01-0.02%

solution of adrenalin or suprarenin. The tampons so soaked must be wrapped in gauze and tied to a string, so that they can subsequently be removed safely. (If in horses or cattle both nares have to be closed by tampons, it is necessary to make a preliminary tracheotomy.) Adrenalin may be injected subcutaneously in doses of 15 to 75 drops per 200 pd. weight. Sometimes ergotin given subcutaneously is very serviceable. In a case of Pease obstinate hemorrhage from the nose was controlled by the intravenous injection of a 2% solution of gelatin in a 0.4% solution of sodium chloride. Hemorrhages from varicose veins or from ulcers may be stopped by cauterization with silver nitrate or by the actual cautery. In very restless horses, narcosis may become necessary in order to secure the arrestment of hemorrhage.

Thrombosis of the nasal veins may occur in passive congestion, in marasmus, occasionally also in phlebitis (Zschokke). It is of some clinical importance, since occasionally it may be confounded with glanders. Corresponding to the thrombi one sees on the mucosa of the septum yellowish or yellowish gray nodules or narrow ridges, the former sometimes arranged like a rosary. The mucosa in their neighborhood appears dark red. The appearance of the nodules, the absence of ulcerations and the lack of swelling of the submaxillary lymph glands are sufficiently characteristic to exclude glanders.

Zschokke saw **abnormally intense congestion in the veins of the nasal septum**, causing nasal stenosis in a horse in the course of thrombotic colic. The horse was breathing with difficulty through the nose and also through the opened mouth and made a strong snuffing noise. The nasal mucosa was intensely cyanotic, and foamy blood was oozing out of both nostrils.

2. Nasal Catarrh. Catarrhus narium.

(*Rhinitis catarrhalis.*)

Etiology. One of the most frequent causes of acute nasal catarrh is cold, which is apt to occur particularly in spring and fall, in cold, foggy, damp weather, and more especially in sudden changes of temperature. The inhalation of air which is contaminated with irritating substances or, in badly ventilated barns, with ammonia, often stands in causal relation. The same is true of excessively warm air and, in case of fire, the inhaled smoke and soot are irritating to the nasal mucosa. Further causes are found in the dust of the street and of the feed, also in mould. Stazzi found, in two dogs, chronic nasal catarrh to be due to Aspergillus fumigatus, and Zürn found Mucor racemosus in the glistening discharge from the nose of a sheep. Further, the inhalation of pollen may give rise to multiple cases of nasal catarrh (see hay fever, page 5).

Foreign bodies introduced into the nasal cavities intentionally or accidentally may cause catarrh or superficial or deep

loss of substance. (Nunn saw a head of wheat drop from the catarrhal nasal cavity of a steer; Parson removed a sponge from the nose of a horse.) The same is true of frequent injury of the nasal mucosa of the horse, which is often produced intentionally in order to mask existing glanders.

Infection appears to play a rôle in some cases, and in that case, nasal catarrh may occur in enzootics. It does not appear unlikely that cold is a predisposing cause in lowering the resisting power of the nasal mucosa.

Nasal catarrh occurs secondarily in some general acute infectious diseases and in other inflammatory conditions of the respiratory organs; it also usually accompanies catarrh of the pharynx and larynx. Inflammatory processes of parts in the immediate neighborhood of the nasal cavities, especially in the bones, teeth, and alveoli of the teeth, usually extend into the nasal mucosa.

Chronic nasal catarrh develops from the acute form only very exceptionally; it is, as a rule, seen as a secondary affection to disease in the immediate neighborhood or in other portions of the respiratory tract. The most common affections leading secondarily to chronic nasal catarrh are: glanders, neoplasms, animal parasites (Pentastomum, Œstrus), catarrh of the accessory cavities, rachitis, chronic bronchial catarrh, pulmonary tuberculosis, lungworm disease, etc.

Symptoms. Acute nasal catarrh usually begins with local symptoms unless it be due to a general infection. One rarely sees a prodromal depression of the sensorium, malaise and moderate elevation of temperature. Smaller animals generally sneeze a good deal in the beginning, shake their heads and rub their noses against their feet or against the floor, while larger animals snort often. The mucosa appears intensely reddened and feels warm. Soon there is a discharge, usually from both nostrils. It is at first perfectly clear, thin fluid, watery; soon, however, it becomes tenacious, glassy, somewhat turbid, at the same time more abundant. Finally, in consequence of the admixture of numerous pus corpuscles, the secretion becomes purulent and less abundant; it dries at the margins of the nostrils and there forms crusts. Later on the discharge again becomes clear and then ceases. The mucosa, which is at first dry, gradually becomes shining. It retains its intense red color during the whole time and becomes more or less swollen; sometimes in the larger animals, usually in the smaller ones, respiration becomes snorting and sniffling. In dogs asthmatic attacks occasionally occur, due to a periodic accumulation of mucus between the turbinated bones (Liénaux). Sometimes rattling nasal sounds are heard. Exceptionally roundish, superficial erosions are formed on the mucosa. The intensely red base of these erosions soon becomes covered by new epithelium.

In rare cases there are formed thin-walled vesicles of millet to lentil-size (so-called "Blatterdruse," rhinitis phlyctænulosa). The process which leads to the formation of such vesicles is a rapid mucoid degeneration of epithelial cells or the accumulation of a serous exudate under the epithelial covering, which elevates it in spots from the underlying tissue. The vesicles later on dry and form crusts which are shed during the rapid regeneration of the epithelium.

One of the most common complications is acute conjunctivitis, manifesting itself by redness and swelling of the conjunctivæ, photophobia, and increased lacrimation. Some patients show difficulty in deglutition, also acute laryngitis, as a complication. The submaxillary lymph glands are sometimes slightly swollen and somewhat tender. The swelling soon disappears with the cessation of the nasal discharge.

Hay fever (pollen disease) is a severe coryza which appears after the inhalation of the pollen of certain gramineae, and is associated with conjunctival catarrh, redness and swelling of the region around eye and nose, sometimes even with attacks of asthma. A particular sensitiveness to pollen-substance is observed only in man. While it may produce nasal catarrh in animals, it is not certain whether a special sensitiveness is present. Walther (B. t. W., 1911, 818) diagnosed an enzootic catarrh of the upper air passages as hay fever, which he observed among horses at the time when the grass was in bloom.

The course of the disease is usually favorable. After the secretion has become purulent, the irritation of the mucosa ceases, the discharge rapidly diminishes, and recovery is complete within one or one and a half weeks. Only in short-nosed dogs, a tendency to a chronic course is observed at times.

Chronic nasal catarrh sometimes begins with the symptoms of acute nasal catarrh, but in other cases the affection develops with chronic symptoms from the start.

The nasal discharge, which always exists, varies in composition and in quantity from case to case and even from time to time in the same animal. It is sometimes more mucoid, tenacious and glassy, sometimes more purulent; it may even be bloody or ichorous and fetid. It is usually not abundant, disappears from time to time entirely, to become again suddenly profuse, and is discharged especially after exercise, sometimes also on lowering of the head (rhinorrhœa, blenorrhœa nasalis). The secretion partly dries at the margins of the nares, it partly runs over the upper lip and leads to the formation of unpigmented streaks in consequence of maceration of the skin. A part of the secretion dries on the margins of the nares, forming crusts; the other portion runs down over the upper lip where it macerates the skin and leads to the formation of unpigmented striae or grooves. The submaxillary glands are usually somewhat swollen and indurated, but do not become adherent to the surrounding tissue.

The mucosa appears bluish or brownish-red, or sometimes grayish-red, evenly swollen or uneven (rhinitis prolifera, S. hypertro). Cicatrices of variable type may also be formed. Catarrhal ulcerations as described above occur more frequently and in larger numbers than in acute catarrh. The swelling of

the mucosa may bring about difficulty in respiration with snorting and sniffling. Stenosis of the nasal cavities occasionally is brought about by masses of exudate which accumulate between the turbinated bones (rhinitis concharum). In such cases thinning and deviation of the nasal septum may occur. The accumulation of masses of secretion and the asthmatic attacks caused by them are seen most frequently in dogs with short noses, and especially during or after exercise. When these attacks, which last only a few seconds, take place, the animals suddenly stand still, the fore-feet spread apart, their inspirations are snoring and accompanied by visible thoracic movements, as though a foreign body had become lodged in the nasal cavity; their glance is anxious (Hébrant & Hermans).

Typical ozena ("stink-nose") is the name given to an atrophic rhinitis occurring in man, in the course of which the mucosa, including its glands, and later the submucosa and the turbinates gradually become atrophied; instead of the normal secretion, a varnish-like deposit forms on the mucosa, and yellowish-brown dry crusts are attached firmly, underneath which the secretions putrefy. In consequence, the exhaled air and the nasal discharge, which is purulent and mixed with greenish clumps, acquire a disagreeable foul odor which resembles that of sweaty feet. Ozena in a wider meaning of the term includes all cases of chronic nasal catarrh, in which the secretions and the exhaled air have a persistent foul odor. Hébrant & Hermans (Ann. 1908, 25) report a case of chronic nasal catarrh in a dog, which resembled the typical form of ozena. Portions of the bony nose, the ethmoid bone and of the bony walls of the nasal accessory sinuses became necrotic and were cast off.

The course of chronic nasal catarrh is protracted, and often the affection cannot be removed entirely. The process shows a tendency to extend to the neighboring organs, in horses to the maxillary sinus or to the air-sac, in dogs, especially in short-nosed animals, to the frontal sinus. Cats sometimes develop a chronic nasal catarrh of unknown origin, which leads to enlargement of the nasal bones, thickening of the skin of the nose and to swelling of the submaxillary glands. In feeble sheep, particularly in lambs, wetting or cold often lead to chronic nasal catarrh with severe local symptoms; it is usually followed by obstinate catarrh of the conjunctivæ and of the lower respiratory passages, which exhaust the animals greatly. It is a question whether this affection in some manner stands in relation to the chronic form of hemorrhagic septicemia.

Diagnosis is usually easy. The decision as to whether catarrh is primary or secondary is, however, more difficult. Since it may be of great importance to ascertain the exact nature of the affection, artificial illumination of the nasal cavities is indicated eventually by the aid of a rhinolaryngoscope. In certain cases the microscopical examination of the nasal discharges is necessary, and trephining of the nasal cavity may be required. All associated symptoms need consideration.

In horses it is necessary to exclude glanders, which is characterized by the peculiar nodules and ulcers, and by the condition of the submaxillary glands (see Vol. I). In the absence

of well defined symptoms, only the mallein test and the sero-diagnostic methods (agglutination, complement-fixation, precipitation) can give definite information. In cases in which, for a time, strangles presents only symptoms of acute nasal catarrh, the subsequent characteristic involvement of the submaxillary glands will decide the diagnosis. Yet, throughout its course, strangles may assume the clinical picture of acute nasal catarrh, and in such cases the specific nature of the disease is recognized only by the fact that other animals of the same herd acquire the typical disease (see Volume I). Specific nasal catarrh must be thought of also in other species of animals. Empyema of the accessory nasal cavities (maxillary sinus, frontal sinus) is to be considered especially in those cases in which the discharge is copious or unilateral, increasing when the head is held low or during motion. In these cases the changes in the nasal mucosa are slight, but the region of one of the accessory sinuses is sensitive to pressure, the bony wall may bulge out, and an exploratory operation of trephining will decide the question of diagnosis. Percussion does not often give reliable results. The differentiation of nasal catarrh and empyema of the guttural pouch in horses is possible by methods similar to those employed in differentiating catarrh of the accessory sinuses.

Treatment. Acute nasal catarrh usually ends in recovery without any treatment. It is sufficient to protect the sick animals against drafts, to keep them in a moderately warm place, and to give them feed free from dust. In severe cases with higher fever, with abundant discharge and snorting respirations, it is well to irrigate the nasal cavities with lukewarm water, or better, with lukewarm physiological salt solution containing one per cent of bicarbonate of soda. If the mucous membranes are very irritable, the irrigating fluid may be diluted with equal parts of milk, or the first irrigation is followed by one containing oily remedies (Ol. amygdal. with 1% of menthol; thymol, eucalyptol, peruol, creosote, guaiacol), which may also be applied with a suitable nebulizer. In horses these oily substances can be applied directly to the mucous membranes of the nose by means of an applicator armed with a pledget of absorbent cotton. Suitable astringent solutions for irrigation are, 1% alum solution or 0.2-0.5% tannin solution; for disinfectants, 2 to 3% boric acid or borax solution, 1% creolin, 0.1 to 0.5% solution of permangauate of potassium. These solutions may render good services if administered with the nebulizer. If the discharge is very copious, douches with 1 to 2% solution of sulphate of zinc, or inhalations of oil of turpentine (1 to 5% evaporated over hot water) are of use. In case the mucous membrane is greatly swollen and the nasal passages are contracted on that account, the direct application of 0.01% solution of adrenalin or of suprarenin should be considered; small amounts of these solutions may be employed for irrigation.

Crusts adherent to the margins of the nares should be removed with warm water and the place should then be covered with fat or vaseline.

Chronic nasal catarrh requires local treatment with inhalation and irrigation of the nasal cavities.

In larger animals **inhalation** is brought about by placing under the nares of the patient a vessel filled with hot water and by wrapping a large cloth around the head of the animal and the vessel. If a horse should be frightened by this manipulation, the lower part of its head is placed into a feed sack, fastened to the head; the bottom of the feed sack is removed and the former connected with the vessel with hot water (Johne). Smaller animals may be made to inhale in a similar manner, or by the aid of a Siegel, a Bulling or a Wasmuth apparatus. The use of these apparatuses appears less serviceable, however, since the greater portion of the sprayed fluid becomes deposited in the lower portions of the nasal cavities (Poeschel, Freund).

Much better results are obtained in all animals from **irrigation of the nasal cavities** with the aid of a syringe connected with a proper rubber tube, perforated in several places at its free end. This tube is introduced high up into the nares and the fluid is injected under moderate pressure. A catheter with lateral perforations connected with an elevated irrigator may also be used. A Frick or a Bayer-Kieselbach spray apparatus which may be introduced into the nares of a horse without difficulty is also quite serviceable.

In smaller animals it is very difficult or impossible to introduce even quite a thin rubber tube or catheter, it is therefore better to fit an olive-shaped tip to a syringe or other apparatus, which has a central bore and is introduced just into the opening of the nose, which is thereby closed during the injection. The irrigating fluid may be injected with syringes with fine and soft tips or, in birds, by means of a glass tube formed like an eye dropper, the fine point of which is bent at an angle.

However, in chronic catarrh of the uppermost portion of the nose these methods are insufficient, and here as well as in necrosis of the turbinated bones there is no other procedure left but trephining of the nasal cavity and irrigation from above with disinfectant and astringent fluids.

Internal medication appears superfluous; sulphate of atropine may perhaps be used in case of abundant secretion. In secondary nasal catarrh the primary affection must be subjected to the proper treatment, which is usually surgical.

Nasal Catarrh with Cutaneous Desquamation in the Neighborhood of the Nose in Horses. This disease, occurring frequently among horses, is probably due to noxious feed, in such a manner that certain irritating substances of such food, during ingestion, come in contact with the skin in the neighborhood of the nose, the mouth and the mucosa of the nose. Varying with the nature of the feed either the changes of the mucosa of the muzzle are more marked or, on the contrary, those of the nose. An origin as indicated above was established by Oekrész who found fifty-eight out of sixty-four horses of one owner affected seven days after they had been fed with chaff from a certain source. The disease did not spread to other horses. Aside from the changes of catarrh of the mouth and nose, desquamation of the skin was seen at the lower part of the head, the pigmented epidermis was shed in thin, but fairly large, lamellæ and flakes, and the skin assumed a peculiarly ragged appearance. Change of feed in such cases, supported by the usual therapeutic applications to the skin of borovaselin, brings about recovery within a few days.

Benign Infectious Nasal Catarrh in Cattle. This disease, by Dieckerhoff also called ephemerous infectious nasal catarrh, is contagious in nature and is usually observed in adult cattle, only exceptionally in calves. It often occurs as a stable enzootic, frequently, however, also sporadically. According to Dieckerhoff, the period of incubation is, as a rule, only two days.

The relation of this disease to malignant catarrhal fever or croup of cattle (see Vol. I) and to infectious catarrh of the upper respiratory passages in cattle (see catarrh of the larynx and bronchi) is still to be established by further observations.

The first symptoms consist in decrease or even in complete lack of appetite, also weakness and a more or less febrile temperature, which, in severe cases, rises up to 40° C. and higher. In milch cows there is a decrease of milk secretion, lacrimation, swelling of the eyelids, a thin mucoid secretion from the nose, reddening of the conjunctivæ and of the nasal mucosa.

The course is acute and recovery occurs in adult cattle generally after one to one and a half days, more rarely after two to three days. Dieckerhoff observed a fatal case in a calf.

Treatment consists in a proper regulation of the diet (good hay and bran mash).

3. Contagious Nasal Catarrh of Birds. Coryza avium contagiosa.

(*Ansteckender Schnupfen der Voegel* [German]; *Coryza contagieux, morve ou roupie des poules* [French].)

Contagious nasal catarrh of fowls is an acute infectious disease characterized anatomically by a catarrhal inflammation of the mucous membranes of the head.

Occurrence. The disease occurs preferably among young fowls, especially chickens, during damp, cold weather, in fall or spring. It is epizootic in character and often causes the death of many birds. Its economic importance depends both upon the great mortality and upon the fact that affected adult hens are unfavorably influenced as to their egg-laying capacity.

Etiology. Contagious nasal catarrh of fowls is due to an infection. This is shown not only by the epizootic occurrence but also by inoculation experiments (authors' observations), and by the observation that the disease is often imported through newly bought fowls (Ammenschlaeger, Feld). The nature of the infectious virus has not been determined exactly, but it is contained in the secretions of the mucosa of the head.

L. Gallez and later on Gratia & Liénaux found in the mucous secretion from the nose of sick chickens an organism similar to the diphtheria bacillus of Klebs-Loeffler. Gallez believed these bacilli to be an attenuated type of the diphtheria bacillus, while Gratia & Liénaux consider it doubtful whether the organism is an attenuated diphtheria

bacillus or a pseudodiphtheria bacillus. Deich demonstrated spirilla-like organisms and also short bacilli rounded at both ends and occurring in groups.

The disease has formerly, but also again lately, been considered as a special clinical variety of fowl diphtheria (Colin, Schrevens, Gallez, Klee) due to the bacillus of fowl diphtheria.

Natural infection takes place by scattering the infected nasal secretion by movements of the head and in sneezing of the sick birds and by the ingestion of these secretions with contaminated food or water by healthy birds. Healthy flocks usually become infected by the importation of infected birds. The disease may, however, be spread just as in fowl diphtheria, by free flying birds coming from a distance.

Predisposing causes seem to be of importance in causing the disease. Damp and cold weather in the fall and spring, the sojourn in cold and drafty places, may assist in bringing about the infection.

Susceptibility. Young or debilitated birds, especially young chickens, are affected most commonly. In some epizootics birds are affected without reference to age.

Anatomical Changes. Aside from emaciation and anemia a glassy or purulent mucus is found in the conjunctival sac, in the nasal cavities and in the pharynx. The mucosa of these parts is reddened.

Symptoms. The disease begins with malaise. The sick animals separate themselves from others and sit quietly with ruffled feathers and drooped wings. The appetite is decreased after one or two days, lacrimation occurs, also frequent sneezing and shaking of the head. At first watery, later on tenacious, whitish to yellowish masses are discharged from the nose. Such secretions are discharged from both nasal openings, and they dry there to yellowish crusts which close up the openings. The birds breathe through the open bill or respiration is rattling or sniffling. The eyes are kept closed and the lids become matted together by masses of secretion; in the conjunctival sacs a whitish, tenacious, however not croupous, exudate collects which makes the eyelids protrude. The cornea appears cloudy. On account of the matting together of the eyelids the sick birds are unable to take up their food and they may actually starve to death. The infra-orbital cell, which is filled with masses of exudate, sometimes protrudes in the shape of a swelling or tumor. In the pharynx we find a muco-serous exudate and intense reddening of the mucosa. Swallowing may become difficult. The birds frequently succumb with rapid emaciation.

Haubold saw in goose-fattening establishments annually numerous cases of a disease resembling contagious nasal catarrh and leading to

serious economic losses. After an incubation period of eight to sixteen days the birds showed reddening of the bill and a mucous nasal secretion which easily dried into crusts. The birds frequently scratched their bills, dipped them often into water, curved their necks occasionally or bent their heads toward the back, shook them, cried out from time to time, turned around in a circle, "turning disease" of geese, and finally fell down almost unconscious, soon to get up again. Beginning with the third week there is diminution of appetite. Sometimes 10 to 15 per cent of all birds succumb to the disease. Other observers claim that it is incurable. It appears that it has been imported into Germany from Russia. Schreiber claims to have found as the cause of the disease a micrococcus, and its toxins are said to affect the cerebellum.

Haubold believes that the affection is an infectious cerebrospinal meningitis.

Course and Prognosis. The disease spreads rapidly among the birds of one establishment and leads to death in some epizootics after only one to three days; generally, however, the course lasts several days, up to three and six weeks. Most young birds die from the disease (sometimes there are 95 per cent of fatal cases); in adult birds the disease frequently ends in recovery.

Diagnosis. The clinical diagnosis is based on the contagious nature of the disease and the mucopurulent character of the catarrh of the mucous membranes of the head. Deposits upon the nasal mucosa are only formed by dried flocculi of fibrin and can easily be removed, whereupon the intact mucous membrane is exposed. Simple nasal catarrh, which under favorable hygienic conditions may also occur among a number of birds, may be distinguished by the fact that it does not spread widely, that the nasal secretion remains thin, fluid, and that the affection of the conjunctivæ and pharyngeal mucosa, which, in rare cases, may coexist, remains moderate in degree. In fowl diphtheria pseudomembranes are found on the mucosæ and also on the skin.

After removal, superficial or deeply penetrating losses of substance remain. Since fowl diphtheria, especially in young birds, may manifest itself only in a catarrhal condition of the mucous membrane of the head, and since the diphtheric deposits, particularly when located in the larynx and trachea, are hard to see or entirely invisible even upon careful inspection, a positive differentiation is not always possible in individual cases. Under such conditions, one should reserve a diagnosis until other cases develop, especially if the purely catarrhal type of fowl diphtheria, by its tendency to form desquamations of the skin of the head in the form of small scales or thick grayish-white crusts, does not permit the elimination of contagious nasal catarrh.

Treatment. Zürn recommends the inhalation of vapors of tar; Azary, irrigation of the nasal cavities with a 3-5 per cent solution of chlorate of potash, or a 5 per cent solution of borax; Klee advocates irrigation of the nasal cavities with a 2 per cent solution of sulphate of copper. The same solution may be used to brush the conjunctivæ and the mucosa of the pharynx. Vallillo had good results from the use of a 3% solution of zinc chloride and a 3% solution of tannin and boric acid. One should also see that the masses of secretion agglutinating the eyes and the nostrils are removed from time to time. The infra-orbital cell, if swollen, must early be laid open by an incision and must be irrigated for several days after the removal of the exudation. One must also attend to artificial feeding of young birds whose eyes have become closed and who consequently cannot feed properly. Feld recommends that white bread soaked in water or milk be introduced into the bill of such patients. While the disease lasts the animals should be kept in warm places.

Prophylaxis. Since treatment of the disease frequently is without results it is advisable to limit the spread of the disease by isolation of the sick, or better still, of the healthy birds, and by cleaning and disinfecting the coops and runs. The spread of the disease may sometimes be checked by the immediate slaughter of all the sick birds (Ammerschlaeger). The importation of the disease may be prevented by careful examination of newly bought birds and by keeping them in quarantine for some time.

4. Infectious Nasal Catarrh of Swine. Rhinitis infectiosa suum.

(*Schnüffelkrankheit, Bösartiger Nasenkatarrh der Schweine* [Anacker] [German]; *Rhinitis infectiosa* [Imminger].)

Infectious nasal catarrh of swine is an enzootic, infectious disease of young pigs, caused by the bacillus pyocyaneus, characterized anatomically by a hemorrhagic inflammation of the nasal and ethmoid mucosa and by hemorrhagic meningitis.

Historical. The disease has been known in Germany for a long time under the name of "snuffling disease"; it was believed to be a rachitic affection of the bones of the face or osteomalacia (see Vol. I) (Haubold, Harms, Wulff, Ostertag); others considered it a specific form of rhinitis (Schneider, Imminger); while still others (Anacker) thought that it was identical with malignant catarrhal fever of cattle or a form of scorbutus (Hering). Since nasal stenosis due to tuberculous or actinomycotic changes has also been included under the term of "snuffling disease" Anacker, and particularly Friedberger & Fröhner, have justly advocated to abandon this name entirely.

Imminger recognized the infectious nature of the disease in 1890 and Koske cleared up its etiology in all of its main points in 1906.

Aside from these authors, Anacker, Schneider and others have furnished contributions to the symptomatology and pathologic anatomy of the disease.

Occurrence. Infectious rhinitis occurs during all seasons enzootically among pigs three to six months old; older hogs are affected only rarely. Wooden pig-pens appear to be particularly favorable to the appearance and to the spread of the disease; other observers claim that a rough stone floor favors the appearance of the affection. So far, only German veterinarians have reported on the appearance of this disease.

Etiology. Bacillus pyocyaneus is a usually small, slender, non-motile rod with rounded ends and with a single flagellum at the posterior pole; its length is between 0.6 to 6 μ and its shape is likewise very variable. It does not form spores. The bacilli have been found in the ethmoidal mucosa and in the brain of sick animals. They are easily stained with the watery anilin stains, but they are decolorized by Gram's method.

Cultural Properties. In a gelatin *stab* culture the bacilli develop almost exclusively in the upper part of the *stab* canal, to form first a depressed growth on the surface, greenish and fluorescent in color. Liquefaction spreads downward and the growth collects at the bottom of the liquefied gelatin as a slimy mass; the surface becomes covered with a greenish-yellow growth. On gelatin plates there are first formed small, roundish, yellowish-white colonies growing rapidly in size, their center darker yellowish and the periphery greenish fluorescent. On agar, there is formed a fairly thick, grayish-yellow covering, which at room temperature later on becomes deep dark green; the cultures emanate an aromatic odor resembling jasmin. On potatoes the growth is at first reddish-yellow and changes from the second day into green. Bouillon becomes after some time uniformly cloudy and greenish in the upper strata. Milk is coagulated.

Pathogenicity. Small laboratory animals (mice, guinea pigs) die in 2 to 5 days after a subcutaneous injection of the culture and in 36 hours following intraperitoneal injection. Inoculation of the culture into the mucous membrane of the ethymoid bone in pigs after trephining the nasal cavity, produces symptoms and anatomical changes similar to those observed in natural infection. According to the amount of culture material injected the animals die in from 24 hours to 8 to 10 days. After intramuscular injection, the animals die in 27 to 30 days. Intravenous injections only cause a temporary rise in body temperature. Leusen doubts that Bacillus pyocyaneus can produce a contagious rhinitis.

Infection takes place through the nose. Since B. pyocyaneus is widely distributed in nature, and since it can be found in large numbers in manure, straw, and filthy water, they easily gain access to the nasal cavity when the pigs are rooting. The inoculation experiments reported by Koske seem to indicate that

other modes of infection are also possible. In infected herds, the disease is spread by the nasal secretions. Young animals are especially susceptible.

Pathogenesis. After gaining access to the nasal mucosa, the bacilli multiply rapidly and soon get into the upper portions of the nasal cavities and into the ethmoid bone. They form powerful toxins and these produce, in the nasal mucosa and particularly in that of the ethmoid bone, a hemorrhagic inflammation which subsequently spreads to the meninges. Bacterial toxins, absorbed into the blood, soon cause a general elevation of temperature and petechiæ and ecchymoses in various portions of the body. Koske showed experimentally that the toxins of the bacillus pyocyaneus can by themselves produce a typical picture of the disease.

Anatomical Changes. The nasal mucosa is deep purplish red and the intensity of the redness is, according to Imminger, somewhat decreased upward. If the disease takes a short course one finds blood coagula in the ethmoidal cells; if the course has been more protracted a hemorrhagic purulent exudate is evident; there may be deviation and atrophy of the bony lamella. The frontal sinuses usually contain a serous fluid. Hemorrhages are seen under the periosteum of the ethmoid and vomer, as well as in the sheaths of the olfactory and optic nerves. The congested vessels of the meninges show blood extravasation; blood is also seen between the convolutions of the brain, sometimes in the shape of blood coagula. The substance of the brain appears edematous and the ventricles contain a red-tinged though clear fluid. Sometimes blood coagula are deposited on the choroid plexuses. Koske found a turbid, reddish fluid in very small amounts in the peritoneal cavity, here and there diffuse reddening of the intestinal mucosa, streaky reddening of the cortical portion of the kidney, subserous hemorrhages and parenchymatous degeneration of the internal organs.

Symptoms. The disease is ushered in by a febrile elevation of temperature up to 41° and above and a diminution of appetite. They early show a certain degree of anxiety and excitement and make a peculiar blowing noise, as if they wanted to expel a foreign body which had gained entrance into the nose. They occasionally rub their snouts on their feed troughs or on the walls of the pens. Usually on the second day there is bleeding from the nose upon sneezing, and there appears a reddish, slimy, later on and in more protracted cases, a purulent nasal discharge; according to Anacker, even ichorous secretion flows from the nose in some cases. Breathing becomes more and more forced and snorting. If the nasal mucosa is much affected, there may be swelling of the region of the nose and of the submaxillary glands. In the further course, generally after one to

three days, marked cerebral symptoms become manifest, there is restlessness, which may increase to maniacal excitement and to attempts to scale the walls of the pens; also convulsions. The excitement is followed by coma and the animals lie apathetically in the straw.

Course and Prognosis. The disease generally takes an acute course and frequently ends fatally within from three to six days, under increase of the disturbances in respiration and of the general depression. In some epidemics all the animals die and it becomes necessary to slaughter the exposed animals in time before they show signs of the disease. Rarely do cases terminate fatally within one to two days. More frequently a chronic course is observed, the first tempestuous symptoms become milder, the appetite, however, remains poor or variable, there may be slight epistaxis at intervals and mild convulsions. The nutrition of the patients suffers gradually and the animals die from inanition after several weeks or months, unless they have previously been killed. Recovery is rare; different epidemics, however, vary greatly in this respect.

Diagnosis. The acute onset with high fever, the intense inflammation of the nasal mucosa and of the brain without protrusion of the bones of the face, furnish sufficient data for a diagnosis. Rachitic or osteomalatic extension of the facial bones is characterized by an afebrile course and by the marked disorder of the facial portion of the head with simultaneous protrusion of the hard palate toward the buccal cavity; there is in this affection a nasal catarrh only after a prolonged course and after a nasal stenosis has been existing for some time. Hóg cholera, in its most acute form, might be confounded with contagious nasal catarrh because it is sometimes accompanied by epistaxis and by symptoms on the part of the brain. The absence of anatomical changes in the nose, and the changes found in the intestinal tract upon post-mortem examination, should prevent an error in diagnosis.

Treatment. This is frequently without success. It appears therefore best to slaughter most of the sick animals. If the symptoms are mild from the start, treatment should however be instituted. Imminger was successful in two cases with instillations of a 1% solution of corrosive sublimate, 1 tablespoonful into each nostril every one to two hours. One may also try irrigation of the nasal cavities with corrosive sublimate or another disinfectant solution. (See page 527.) Applications of a few drops of suprarenin or adrenalin, in 1% solution, may be tried, or this solution may be diluted with physiologic salt solution and used for irrigating the nasal cavity.

Prophylaxis. If the disease appears in a herd, it is well to separate the healthy animals and instill some corrosive subli-

mate or other disinfectant solution into their nares. The infected pens must be well disinfected and kept clean.

Chronic Purulent Hemorrhagic Rhinitis with Atrophy of the Turbilated Bones of the Nose in Young Pigs. This disease occurs in young pigs up to six months of age in certain regions of Germany (Naussau) and of Denmark (Jensen). It is said to develop as a result of a congenital predisposition in animals with a relatively short snout and greatly atrophied turbinated and ethymoid bones, because of the fact that foreign bodies that have gained access to the unusually roomy nasal cavity cannot be expelled by blowing, as under such conditions the force of the air current is much reduced. The irritation of the nasal mucosa by the foreign body and the micro-organisms attached to it, results in a chronic, purulent or hemorrhagic catarrh of the nose.

The symptoms consist of the presence of labored breathing with a snuffling noise, at first only while feeding and when the animal is exercised; later also while the animal is at rest. Further symptoms are an increase in the circumference of the relatively short snout and the middle part of the upper jaw, mucoid or bloody nasal discharge, periodic attacks of suffocation with improvement following hemorrhages from the nose, and a medium degree of emaciation. Such animals either die in a cachetic condition or are slaughtered when improvement has taken place.

For treatment Schneider recommends to try irrigation of the nasal cavity. Under all circumstances, animals inclining to this condition, or animals that have already been infected, should be eliminated from the breeding herd.

5. Contagious Nasal Catarrh of Rabbits. Rhinitis contagiosa cuniculorum.

(*Influenzaartige Kaninchenseuche; Infectiöser oder Bösartiger Schnupfen, Bösartiges Schnupfenfieber, Bösartiges Katarrhalfieber der Kaninchen, Kaninchenstaupe* [German]; *Rhinitis Purulenta.*

Snuffles is a contagious disease of the respiratory organs, usually of an enzootic character and essentially of polybacterial origin.

Historical. This disease, which has long been known under the name of "malignant snuffles," was formerly believed to be a coccidia rhinitis of rabbits (see page 31). The investigations of Beck (1891), Kraus (1897), Roger & Weil (1901), Volk (1902) and Kasparek (1903), have, however, shown that the great majority of cases known under this or similar names is of bacterial origin. Affections similar clinically and anatomically have been observed by Suedmersen (1905), Koppányi (1906), and by Raebiger (1907), but they are due to bacteria of a different kind.

Etiology. The disease is due, in some of the cases at least, to a small bacillus resembling the influenza bacillus. It is a very small, slender, non-motile rod about the size of the fowl-cholera bacterium, Gram negative and does not form spores.

Cultivation. In the presence of oxygen and at blood temperature the bacilli grow on all of the usual culture media. On gelatin plates there are formed after forty-eight hours small granular colonies with a sharp or, according to Kraus, serrated margin; the gelatin does not become liquefied. On the surface of agar there is developed a luxuriant grayish-white, iridescent growth. Milk is not coagulated, indol is not formed. The bacilli described by Beck, Kraus, Volk and Kasparek show some differences, mostly cultural in type, and according to Kasparek this points to the fact that different varieties exist. The variations are, however, not considerable enough to justify a differentiation of the affections caused by the various types.

Suedmersen found a bacillus of the coli group in pleurisy of rabbits which is identical in its main characteristics with bacilli found by Kraus and Tartakowsky in a similar disease of guinea pigs. Raebiger found an ovoid bacterium which corresponds with the Bacillus bipolaris septicus. The Pyobacillus capsulatus cuniculi isolated by Koppányi is distinguished by the fact that it is polymorphous, rather plump and surrounded by a capsule.

The infection is transmitted by the inhalation of particles of nasal secretion that are thrown into the air in the form of a spray during violent exhalation and are either inhaled directly, or with particles of dust. Feed contaminated with nasal secretions also plays an important role in the spread of the disease, as do also the hands of the attendant. Animals of all ages may become infected.

Pathogenesis. After their entrance into the nose, the bacilli produce a violent inflammation of the mucosa of the nose and of the accessory cavities and they also get into the general blood circulation; there then occurs an elevation of the general temperature and in some epizootics, in fact, in most cases, an inflammation of the serous membranes and a pneumonia. The inflammation may spread from the nasal cavities into the deeper portions of the respiratory tract.

Anatomical Changes. The mucosa of the nose and pharynx appears intensely reddened, swollen and covered with purulent material. The accessory nasal cavities, frequently also the bronchi, contain a purulent exudate. In the pleura we find occasionally a serous, stringy or even purulent exudate, and the surface of the lungs is covered with a fibrinous deposit. The pericardium is rarely affected similarly. The lungs often show, aside from compression, atelectatic or bronchopneumonic foci. The specific bacilli are found in large numbers in the exudate of the mucosæ, in the other affected organs and in the blood.

Symptoms. Weakness and depression become apparent after a period of incubation of four to six days (according to

Raebiger, in eight to fourteen days); the nares become moist and there is frequent sneezing. The temperature rises to 40° C. and above, and the appetite decreases. The nasal secretion is at first scanty and watery, or thick mucoid, and wets the hairs in the nasal region, the thorax and the anterior extremities; it becomes more abundant, purulent and milky between the second and fifth days of the disease. Material of this kind is expelled by sneezing and snorting, and the animals rub their noses with their front legs. The appetite diminishes more and more, there is emaciation and debility and also dyspnea and cough.

In pleurisy with pyemic cachexia of rabbits, as described by Koppányi, very acute cases occur leading to death in two to three days, with rapidly increasing dyspnea and great prostration, high temperature, followed by a rapid fall of the temperature to below normal. Then chronic cases are frequently observed with the formation of subcutaneous encapsulated abscesses on various parts of the body or in the internal cavities. After the spontaneous or artificial opening of such abscesses, the animals may recover, or more rarely they may die on account of acute exacerbations. Otitis interna sometimes occurs with oblique holding of the head or acute meningitis or cerebral abscess with symptoms of excitement. Abscess is observed also in other forms of infectious catarrh of rabbits.

Course and Prognosis. In many cases the symptoms are intensified rapidly and the animals die within three to five days (acute type). In other cases, however, the symptoms are less acute from the start and the disease lasts fifteen to eighteen days (subacute form), or it lasts for a long time with mild symptoms (chronic form). All acute cases end fatally; a complete recovery is seen rarely also in the subacute or chronic variety.

Diagnosis. The severe local symptoms accompanied by high fever from the very onset, serve to differentiate this disease from other similar affections. In rhinitis due to coccidiosis, the fever, debility, and difficult respiration are, at least at the beginning, not so severe. A microscopical examination will reveal coccidia in the nasal secretion. The inflammation of the serous membranes is absent. Salivation in rabbits, a special type of vesicular stomatitis, can be differentiated from snuffles by the absence of fever and other general symptoms, by salivation in the absence of the discharge from the nose, and the nodules and vesicles that soon appear on the lips.

Treatment and Prophylaxis. Since irrigation of the nasal cavity (see page527)with 1% creolin or 3% boracic acid solution does not materially influence the course of the disease, the main efforts have to be made in the direction of prophylaxis. This consists in strict separation of the healthy from the affected and the suspected animals; the healthy and the sick animals should be fed and cared for by different persons. If

these precautions are not feasible, all sick animals must be killed at once, at the beginning of the epizootic and their cadavers must be destroyed. The cages and the barn, including the walls, should be thoroughly cleaned and disinfected; newly bought animals should be kept in quarantine and under observation for fourteen days. The prophylactic immune serum of Raebiger did good service in one infected lot and exerted even a certain curative action upon the sick animals.

Infectious Nasal Catarrh of Guinea-Pigs. This disease is similar to the infectious rhinitis of rabbits; it was observed in enzootics by Kaspar & Kern (Cbl. f. Bakt. 1912. LXIII, Orig. 7), who isolated the Micrococcus tetragenus as its causative agent. The affection is probably introduced by carriers of the bacterium, and it is then disseminated by the cocci which are eliminated with the nasal discharge. It was not possible to transmit the disease to animals of other species.

The symptoms of the acute form consist in copious, mucous nasal discharge, frequent sneezing, and severe diarrhea. Death ensues in only a few days with convulsions. In the chronic cases, which develop if the enzootic is protracted, abscesses form in the lymph nodes, under the skin and in the substance of the muscles; there is emaciation, and the disease usually ends in death in the course of a few weeks; recovery takes place rarely, after the abscesses have ruptured.

Contagious Rhinitis in Cats. As in contagious snuffles in rabbits, this is also an enzootic, general infection that localizes particularly in the mucous membranes of the head, the posterior air passages, and the thoracic organs. During an enzootic in Germany, Gaertner found the Bacillus felisepticus (see croupous pneumonia) as the cause, while during an enzootic in Leyden, de Jong found a streptococcus. According to the latter author, the disease was formerly diagnosed as distemper.

The symptoms consist of decreased appetite, frequent sneezing, and a profuse nasal discharge which at the beginning of seromucoid in character and in 2 to 3 days becomes purulent. During respiration, the masses of pus will move back and forth in the nostrils, partly dry up into crusts and cause the animal to breathe through the open mouth. Respiration is labored, often pumping, accelerated, and accompanied by loud wheezing and snoring. Frequently also a weak, empty and painful cough is present. As a rule, a severe conjunctivitis which at first is serous in character, later becoming purulent, is present. The body temperature varies from 39.5° to 41.6° C. At the beginning, the animals appear dull and surly and later become distinctly apathetic. During a protracted course the animals become emaciated, anemic, and weak.

In the cases of Gaertner the disease usually terminated fatally in 3 to 6 days and was usually accompanied by croupous pneumonia. In the case of de Jong, it had a great tendency to terminate favorably, especially when the animals were placed in fresh quarters and well taken care of. Only once was it accompanied by light bronchopneumonia, while more frequently a light fibrinous, pleuritis and peritonitis was present.

6. Croupous Rhinitis. Rhinitis crouposa.

(Nasal Croup.)

Croupous rhinitis is an intensely inflammatory process of the nasal mucosa, usually with the formation of extensive pseudomembranes.

Occurrence. Nasal croup is usually seen as an independent disease among horses, more rarely among cattle. The disease not infrequently appears enzootically among horses, particularly among the animals of a breeding establishment, and is then sometimes associated with follicular inflammation of the nasal mucosa (Roell).

Etiology. The disease sometimes appears after the inhalation of hot air and smoke in conflagrations. In most cases, however, an infection appears to be the causative factor. Roell observed the spread of the disease from sick to healthy horses. Obolenski, Berndt and Grunth observed a contagious spreading of nasal croup among cattle. (See Croup of Cattle, Vol. I.) The suspected infectious virus is not known as yet; in horses, however, the possibility that streptococcus equi is the etiologic factor cannot be entirely discarded. In one of the author's own cases this disease followed after streptococcus mastitis in a mare, and in cases in cows, of which Strebel, Wyssmann, also Zwick & Weichsel, each reported one instance, it was preceded by a parenchymatous mastitis. Reinhardt & Hofherr report the case of a cow ill with mastitis necrotica caused by Bact. coli, in the course of which a croupous-diphtheritic inflammation of the mucous membranes of nose, pharynx and larynx made its appearance.

Nasal croup is seen as a secondary affection in the course of some infectious disease (rinderpest, malignant catarrhal fever, morbus maculosus, strangles, etc.).

Symptoms. The disease is initiated with the symptoms of a severe acute nasal catarrh, followed soon by the formation of gray or reddish-gray thick pseudomembranes upon the intensely red and swollen mucosa. These pseudomembranous deposits are either confined to smaller areas or they form larger, more extensive, continuous patches. They can be easily detached and after this has been done spots of mucosa are exposed to view which are void of epithelium, granular, intensely red and easily bleeding. After a few days the pseudomembranes become detached spontaneously. The epithelial covering is then replaced, the mucosa becomes gradually paler, and recovery takes place without leaving any permanent changes.

There is a yellowish, tenacious nasal secretion which is later mixed with shreds of pseudomembranes. Respiration is forced and snorting in severe cases. The soft parts of the nasal region,

the submaxillary lymph glands and the afferent lymph vessels are more or less swollen and tender. Very rarely nodules and ulcers are seen in the neighborhood of the nares similar to those in follicular inflammation of the nasal mucosa. In the case of Zwick & Weichsel, cited above, membranous deposits formed on the muzzle, which left ulcerations behind, on being removed. The body temperature is markedly elevated up to the shedding of the pseudomembranes.

The course is usually favorable; recovery occurs in about a week, provided that the other portions of the respiratory tract have not been affected.

Treatment. Treatment is similar to that employed in acute nasal catarrh. The shedding of the pseudomembranes may be hastened by irrigation of the nose with a 1 or 2% solution of bicarbonate of sodium. Since the disease is often contagious, it is advisable to isolate the sick animals.

Diphtheritic Inflammation of the Nasal Mucosa. It occurs very rarely as a primary disease after traumatic, chemical, thermal insults or after an infection (bacillus necrophorus); as a rule there are other underlying primary affections (morbus maculosus, acute glanders, also strangles, malignant catarrhal fever). Either moist or mostly dry deposits, especially in glanders, adhering firmly to the base, are formed and upon degeneration are partly changed to a yellow cheesy mass, which sometimes exhibit ulcerated depressions. After the shedding of these masses, deeply penetrating dark red or grayish-red, uneven ulcers with more or less elevated margins, become visible, around which the mucosa is swollen, reddened, of a gelatinous character and sharply defined against the necrotic tissue. Respiration is labored and accompanied by a wheezing sound. A mucopurulent nasal discharge is present. Acute nasal diphtheroid changes accompanying glanders can usually be eliminated by reason of the absence of nodules and ulcers of glanders and the presence of the above mentioned symptoms, which, however, sometimes only becomes possible by an exhaustive bacteriological examination (Rabe, Miessner), or by the application of the mallein tests or blood tests, since extensive diphtheroid changes may hide the changes usually found in glanders. In the interpretation of the results of the blood tests and mallein test, one should not forget that horses with acute glanders will sometimes show no biological reaction for a period of three weeks.

7. Follicular Inflammation of the Nasal Mucosa. Rhinitis follicularis.

(Rhinitis pustulosa; Coryza pustulosa equorum [Kitt].)

Follicular rhinitis is a peculiar inflammation of the nasal

mucosa with nodule formation of the mucous glands, which nodules break down later on. The sebaceous glands of the skin of the region of the nose become similarly affected.

Occurrence. This is a disease of solipeds which occurs enzootically under conditions similar to those which are observed in nasal croup.

Etiology. The disease undoubtedly owes its origin to an infection, as shown by the observations of its contagious nature by Roell and later by Friedberger & Fröhner. Kitt believes that streptococcus equi is the causative factor; Friedberger & Fröhner likewise believe that this is probable.

Symptoms. The disease begins with the symptoms of a violent nasal catarrh and as a rule with fever, whereupon, after two or three days, nodules, of the size of a millet seed, develop on the intensely reddened mucosa of the nasal septum. These nodules are quite numerous and they can be felt easily with the palpating finger. The nodules increase in size, become yellowish and frequently confluent and form a continuous yellowish surface; this disintegrates, becomes pale yellow and leaves after its removal small, roundish, shallow, intensely reddened ulcers with slightly infiltrated margins. The ulcers heal completely within a few days. In most cases similar nodules are developed on the skin of the alæ of the nose, the upper lips and the cheeks. These nodules also become yellow, ulcerate and finally heal without leaving any traces. The lymph vessels, which drain the affected portions, are usually much swollen and can be felt as tender cords leading from the angles of the mouth backward, and also down toward the neck. Sometimes abscesses form in some places. The submaxillary lymph glands are always in a condition of acute swelling. Sometimes one also sees nodules on the generally reddened conjunctiva of the eyes. The disease lasts two to four weeks and generally ends in complete recovery.

Diagnosis. The affection is distinguished from glanders in that the nodules and ulcers appear simultaneously or within a short time over a larger area in great numbers and that they heal without leaving any trace.

Treatment. Irrigation of the nasal cavities with mild disinfectant and astringent fluids (see page 527) and subsequent applications of desiccating ointments to affected cutaneous regions are very serviceable. Also applications of ointments containing mercury, iodoform, creolin, or iodin to the affected cutaneous regions.

Rhinitis bullosa. Under this name Schnuerer described a disease occurring enzootically in army horses at the beginning

of the World War and which was later observed and more accurately described in army horses by other Hungarian and Austrian veterinarians (Sturm, Balla, Toutenui). Toutenui also repeatedly saw it in horses of the rural population of Hungary, while Schneider saw a similar disease in the army horses in Switzerland which has been described as acne nasalis or pseudoglanders.

Etiology. All observers agree that the disease is of bacterial origin since it usually continues to spread, although it has not yet been observed to spread from horse to horse by direct contact. Particularly young and old or greatly emaciated horses are frequently affected with a severe type. According to Sturm, the period of incubation is 2 to 3 days.

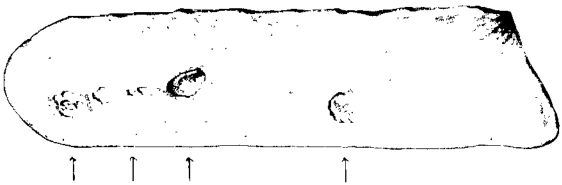


Fig. 101. Ulcers on the mucosa of the nasal septum in rhinitis bullosa. a, Erosions; b, ulcers reaching into the submucosa; c, penetrating to the cartilage; d, perforating the cartilage.

Symptoms. The disease begins with a unilateral or bilateral appearance of roundish, rarely somewhat elongated, hemorrhagic spots the size of a pin-head on the otherwise pale-red mucosa of the nasal septum. At first and in greatest number, such spots will be seen in the lower region of the nasal cavity which is more or less constricted by the bulging of the turbinated bones, but soon these also appear in the upper region, and upon the mucosa of the turbinated bones. According to some observers, cases with only a few hemorrhagic spots are not infrequently encountered. On the other hand, the hemorrhagic spots may be so numerous that the whole mucosa appears as though spattered with blood. After 2 to 4 days, the hemorrhages begin to fade and soon disappear, or, after becoming pale-red in the center, they may be converted into grayish-red or grayish-white spots the size of a lentil with a red margin on the mucosa which may simultaneously show epithelial defects in some places. Occasionally the disease disappears without a trace in the hemorrhagic stage after 5 to 6 days.

In most cases, the hemorrhagic stage is followed by a nodular or vesicular stage in which the grayish-white spots with a

red margin, by gradually increasing in size up to that of a bean, are converted into grayish-white, raised, and somewhat firm nodules around which fresh hemorrhages may occur in the meantime. After 1 to 2 days, the nodules are converted into grayish-yellow flaccid vesicles with a mother-of-pearl luster resembling a drop of mucus. These vesicles are soft and fluctuate upon palpation and burst easily under light pressure, or spontaneously when about a day old, discharging a muco-purulent or more lymph-like content, but on account of their spongy character they still retain their bulging shape for 1 to 2 days longer. Upon pressure with the finger, the contents are sometimes displaced into the adjoining tissue spaces of the mucosa which now become white and permit the formation of several small vesicles. Some of the vesicles may disappear without a trace in 7 days.

The appearance of the vesicles usually leads to the ulcerous stage, since after the disintegration of the wall of the vesicles, crater-like ulcers with a smooth margin and grayish-yellow base, are formed. They may be shallow, but as a rule penetrate deeper, even to the cartilage. They are the size of a copper cent or a little larger, and are surrounded by a red zone. While the shallow rosions may heal in several days without a scar, the deeper ulcers often coalesce to form large ulcerous areas with irregular or ragged, but otherwise slightly elevated, margins. The secretion at the base of the ulcers is always purulent and dried into a crust. Sometimes perforation of the nasal septum takes place, forming holes of varying sizes with a smooth edge and surrounded by a red zone.

The cicatricial stage follows all cases in which a deeply penetrating loss of substance has occurred. The scars are at first grayish-yellow, and project slightly above the surface. In the case of round ulcers they may be slightly radiating and often brownish in color due to the deposition of pigment. In other cases, they are grayish-white or pale-red. The cicatricial contraction produces pit-like scars and deviation of the nasal septum. Very deep losses of substance heal with excessively proliferating granulations. Small perforating ulcers are usually closed again by the healing process, while large ulcers, although growing smaller, do not close.

In the majority of cases other symptoms are lacking. In cases of deeply penetrating losses of substance, thin, dry crusts will be found adhering to the nostrils and breathing is accompanied by wheezing or roaring sound, due to the partial occlusion of the nasal cavity by partly detached crusts. The sublingual lymph glands are usually normal, seldom slightly swollen for a short time and sensitive to touch. At the outset, Sturm sometimes observed a light fever (but little over 38°) without general disturbances or derangement of appetite.

Course. Cases in which only a superficial loss of substance

has occurred will heal in 7 to 14 days, while otherwise 3 to 12 weeks, on the average 6 to 8 weeks, are required for complete recovery. Sometimes permanent stenosis of the nasal cavity with labored, roaring breathing will result.

Diagnosis. The diagnosis is rendered easy by the peculiar symptom complex. The absence of typical nodules and ulcers of glanders, the vesicular origin of the ulcers, the absence of symptoms of irritation in other portions of the nasal mucosa (excepting the red zone around the vesicles and ulcers), the absence of simultaneous swelling of the sublingual lymph glands and of nasal discharge, the absence of fever and other symptoms of disease, and the pronounced benign course and often rapid recovery, serve to differentiate it from nasal glanders. In case of necessity the mallein test and blood tests should clear the point.

Treatment. In severe cases, treatment consists of nasal irrigation with mild disinfectants. Light cases usually recover without treatment. Isolation of the sick animals is indicated in all cases.

8. Tumors in the Nasal Cavities. Tumores narium.

Occurrence. Neoplasms are usually rare in the nasal cavities. Relatively most frequent are polypoid growths (Kärnbach) which arise from a proliferation of all layers of the mucous membrane, and commonly are localized near the opening of the nares and in both nostrils. These tumors are one-third to one inch high and are divided by deep grooves; their surface is irregular, knobby or smooth, their consistency firm or soft. Aside from the hyperplasia of the mucous membrane, the tissue shows much cellular infiltration and not infrequently amyloid degeneration. The growths have been designated as hyperplasia of the nasal mucosa (Siedamgrotzky); as adeno-fibrous hyperplasia (Kitt). Dieckerhoff & Grawitz identified them with rhinoscleroma in man, but this differs essentially from polypoid growths by its specific cause, its structure, its cartilaginous consistency and its localization in the upper portions of the nose. True nasal polypi are less frequent and form sharply circumscribed swellings with smooth, glistening surfaces. Very rare are angioma, lipoma, osteoma, ecchondroma, somewhat more frequent sarcoma and carcinoma; in cattle mucous cysts occur at times.

Tuberculomata occurring but seldom in the nasal cavity of cattle and hogs have a similar clinical significance. They appear in the form of nodules, spread along the surface, are covered with muco-purulent material and contain yellowish spots. They are surrounded by crater-like ulcers with prominently elevated edges and may finally entirely occlude the nasal cavity.

Gerspach saw in a horse tuberculous nodules of pea-size alternating with tuberculous ulcers covered with yellowish, dry adherent crusts. Very rarely actinomycomata are found in

cattle in the shape of strawberry-like masses in the lower portions of the nasal cavities. Extensive cicatricial thickening of the mucosa and deviation of the nasal septum may cause similar symptoms of stenosis as nasal tumors.

Symptoms. A tumor growing in the nose usually makes, in proportion to its size, one, rarely both nasal cavities more narrow, and thereby produces difficulty in respiration, blowing or snoring. According to the degree of stenosis, these symptoms are observed either during exercise or likewise during rest. If the stenosis is produced by a motile neoplasm, dyspnea and the noises accompanying it are observed only occasionally, or the character of the noises changes. Sudden attacks of asphyxia may be caused by non-pediculated trne nasal polypi. If the nasal opening of the affected side is closed the noises cease, while closure of the healthy side increases them and increases dyspnea. Differences in the volume of the air exhaled from either nostril may be ascertained by holding the moistened back of the hand in front of the nose or by placing a mirror in front of them and watching the deposit of dew, respectively, the difference in the size and the time of disappearance of the dewy deposit (Kaernbach). Labial breathing is observed in more severe cases. It disappears, however, on opening the mouth. With the exception of horses, animals breathe either temporarily or permanently through the open mouth and the respiratory noises mentioned above are then not heard. Tumors growing in the lower portions of the nasal cavities are detected by direct inspection and palpation, while those in the upper portions of the nasal cavities are felt upon sounding of the nasal passages.

There is usually a nasal discharge, which is, as a rule, unilateral, mucopurulent, in malignant tumors often fetid, sometimes mixed with fragments of tissues. Angiomas and malignant neoplasms may lead to periodical epistaxis, while tumors of the upper portion of the nasal cavities which extend into the pharynx may cause disturbances of deglutition. In malignant tumors, the configuration of the face is changed not infrequently, the hard palate bulging into the mouth; at times the eyeball protrudes. Chronic swelling of the submaxillary glands on the affected side is usually present, but becomes severe only in cases of malignant and tuberculous tumors.

Treatment. Exceptionally only is any treatment serviceable aside from surgical interference. An angioma situated near the anterior nares may be made to shrink by brushing with a 10% solution of trichloracetic acid, or it may be destroyed by cauterization.

Enzootic Tumors in the Region of the Ethymoid Bone. At present these tumors seem to be limited to Norway and Sweden. On some farms they seem to occur in a large number of cattle

and horses. They develop in relatively old animals. According to the investigations of Stenstroem, Forssell, Magnusson, Horne and Stenersen and Stalfors, the tumors always originate in the region of the ethymoid bone and produce metastasis in the regional lymph glands in only about one-third of the cases. They are of a malignant character and show a great tendency to penetrate the nasal tissue, as the result of which all body cavities may become filled with the tumors in advanced cases. Not seldom a tumor penetrates into the cranial cavity and produces pressure upon the brain. In about two-thirds of the cases the tumors occur unilaterally. Histologically the tumor appears as a sarcoma or a carcinoma, but sometimes as a mixture of mesenchymal and epithelial tumors, while the type may vary even on the same farm. According to Forssell, they resemble infectious granulomata to a certain extent. The cause is assumed to be a specific infection, although bacteriological examination and transmission experiments have been negative.

Enzootic tumors have also been reported from other countries, particularly by Chabert in France, by Loeb in England and America and by Voges in Buenos Aires. Since these tumors occurred in the region of the eye, their relation to those occurring in Sweden cannot be determined.

Symptoms. The symptoms consist of labored breathing, accompanied by a wheezing or roaring noise, hemorrhages from the nose, and foul-smelling mucopurulent nasal discharge. Later there occurs unequal and partial bulging of the bones of the head, protruding of the eyeballs, sometimes swelling of the sublingual and subparotid lymph glands, disturbances of the central nervous system (lethargy, forced movements, attacks of rage) depression of the appetite and fever. Sometimes the nasal discharge and the bulging of the facial bones are absent and only disturbances of the central nervous system are observed. According to Stolfors, the tumor can be palpated in the region of the choanæ and the region of the ethmoid bone (in horses the tumor seldom affects the nasal passages) even before the above symptoms become manifest.

Treatment. The operative removal of the tumors as tried by Vennerholm and Forssell usually is not successful.

9. Animal Parasites in the Nasal and Accessory Cavities.

(a) Œstrus ovis.

(*Bremsenlarvenkrankheit, Œstruslarvenkrankheit, Hornwurmkrankheit, Bremsenschwindel, Schleuderkrankheit* [German]; *Vertige d'oestres, faux tournis* [French].)

The disease caused by the larva of the sheep fly is character-

ized anatomically by a catarrhal inflammation of the nasal and accessory cavities.

Historical. Œstrus larvæ were first observed in the head cavities of sheep by Vallisnieri in 1712; Œstrus ovis became well known mainly through the descriptions of Bracy Clark (1797) and Hertwig (1838).

Occurrence. The disease is found all over the world, excepting Australia, especially in young animals (yearlings).

Etiology. The **larvae of Oestrus (Cephalomyia) ovis** (Linné) are a stage of evolution of the sheep-fly which is 10-12 mm. in length, yellowish gray in color; its wings are striated transversely. The length of the larvæ varies from 2-20 mm., according to the stage of evolution; they are made up of 11 rings, of oval shape, flat on the ventral surface on which fleshy, small protuberances, ending in red thorns, are attached from the second ring down; the dorsal surface is convex. The youngest individuals are white, and transparent, the others yellowish white. The mature ones show a transverse striation of the rings; the anterior end is more elongated than the posterior one. The head ring carries two mouth hooks (Fig.102).

Fig. 102. Larva of *Œstrus ovis* (seen from the posterior surface at the left; seen from the ventral surface to the right).

Evolution of the sheep-fly. The larvae which leave the ova already in the oviduct are deposited by the female flies in the neighborhood of the anterior nares of the sheep, whence they wander into the nasal cavities, *frontal sinuses, ethmoidal cells, and even higher up.* They become adherent to the mucosa and progress in development.

According to Ruppert, the fly deposits its eggs upon the skin of the abdomen, the internal aspect of the hind legs and around the anus. The fly is often found resting upon these parts. The sheep are said to take up the eggs and young larvæ by licking these parts of the body. According to the same authors, the eggs are probably also deposited in the crevices in walls and on shrubs and taken up by the sheep while nosing about. After the larvæ have become mature in the spring at the end of about ten months they leave the host by way of the middle meatus and of the nostrils and reach the pupal stage in moist soil or manure within 24 hours, provided the temperature is above 12° C. According to Ruppert, it takes 2 to 3 days. Varying with the outside temperature, the two-winged gad fly hatches in from 4 to 6 weeks in a warm temperature to from 2 to 3 months in a cooler climate. Soon afterwards the female will be impregnated and begins to deposit larvæ.

The period during which the larvæ are deposited, lasts from the middle of May to the last days of October; in colder regions it is of shorter duration. The sheep-fly remains by preference in cracks and holes in sheep-barns, also in low shrubs near the barns and pastures. From here the fertilized females hunt up the sheep in dry, warm weather, especially during the middle of the day.

Anatomical Changes. The mature larvæ are, as a rule, found in large numbers (according to Zürn, 60 to 80) in the

frontal sinuses and in those at the base of the horns; sometimes, also in the antrum of Highmore and in the nasal cavities. They are strongly adherent to the mucosa which here shows small roundish depressions and in their neighborhood catarrhal changes. Exceptionally the brain and its membranes become invaded and likewise exceptionally the larvæ are found in the pharynx and larynx.

Symptoms. On the approach of the flies the sheep run together, put their heads together or place them on the ground, or hold their noses between their legs. If the larvæ are deposited in spite of this, and after they have begun to creep upward into the nasal cavities, the sheep become very restless, rub their heads on the soil or between their feet, shake themselves, sneeze frequently, and run around restlessly. The restlessness, however, soon disappears and the animals remain quiet for about ten months. The larvæ cause irritation of the mucosæ of the head only after maturity or after their migration, symptoms become manifest only in spring or in early summer. The actual sheep-fly disease usually begins with the mild symptoms of an acute nasal catarrh, which increases in intensity within the next weeks. The first and most constant symptom is nasal discharge, frequently unilateral, at first clear, serous or seromucoid, later purulent; or possibly hemorrhagic. The animals frequently sneeze and blow and throw out an abundant discharge, occasionally also some larvæ. At the same time, the sheep rub their noses on their front legs, on neighboring objects and on the ground, so that the parts around the nose sometimes become denuded and subsequently edematous. From time to time the head is lowered, then suddenly elevated, bent energetically backward or laterally; the gait is staggering and uncertain. Simultaneous catarrh of the conjunctivæ leads to profuse lacrimation. The mature larvæ leave the nasal cavities after one to two weeks, and the nasal and conjunctival catarrh then disappear.

In rare cases the catarrhal symptoms, which have become severe, are complicated by signs of excitement and depression. The sick animals raise their feet high, stagger with the hind legs and repeatedly fall down. There is grating of the teeth, rolling of the eyes, and occasionally forced movements toward one side (Gilis) are observed; also partial or general muscular convulsions. In such cases death usually occurs within five to eight days, exceptionally within three to four days after the first symptoms become manifest.

Diagnosis. The rare cases with forced movements may be confounded with giddiness of sheep. This, however, affects almost exclusively young animals, and forced movements stand in the foreground of the clinical picture, while catarrhal signs are absent. The differential diagnosis, however, meets

with great difficulties and becomes impossible when Œstrus larvæ situated high up lead to nervous symptoms, while symptoms of catarrhal affections of the mucosæ are absent, or if no Œstrus larvæ are expelled. The disease can be differentiated from lungworm disease by the absence of cough, by the occasional presence of Œstrus larvæ in the nasal discharges, while lungworms and their ova are not found.

Treatment. The only rational procedure consists in opening up the affected cavities, removing the larvæ that are present and accessible with a pair of forceps and anesthetizing those remaining with appropriate irrigations. This treatment, however, can hardly be carried out when a large number of animals of a herd are affected and the operative procedure will then perhaps be employed only on the most valuable animals. The treatment sometimes does not have the desired effect and most animals, especially in the presence of severe cerebral symptoms, will, according to Zürn, remain sick and will succumb. Speedy killing of the animals is therefore indicated if the affection is violent, if trepanation cannot be carred out, or if, in spite of trepanation, the symptoms do not ameliorate.

Trephining of the Frontal Sinuses. Since the larvæ are found preferably in the frontal sinuses, these must be laid open in particular. Zürn recommends as the place of trephining the two upper, Moussu, however, the lower angles formed by a line connecting the superciliary ridges with another line drawn at right angles into the middle of the former line. If the condition does not improve after trephining in this manner, it is advisable to remove the horns and thus to lay open the cavities at their base. After the larvæ have been recovered from the opened cavities, these must be irrigated with a fluid noxious to Œstrus larvæ. Serviceable fluids are: Lugol's solution, benzin diluted with water, oil of turpentine, 3% carbolic acid, 95% alcohol, lime water, etc. None of these fluids will kill the very resistant larvæ (Fischer), but they will cause them to contract strongly, so that they will leave their places of attachment and will be expelled from the nose by strong sneezing, or can be removed through the opening made by trephining, especially if this is closed a few minutes after the injection.

The insufflation into the nose of tobacco, snuff, heleborus, all of which cause sneezing, the inhalation of irritating smoke, irrigation with various fluids (aside from those already named, vinegar or salt water), is frequently recommended. Considering the great resistance of the larvæ and their place of location, one cannot expect much success from such procedures.

Prophylaxis encounters very great difficulties. It is advisable to destroy the larvae. Sheep flies may be driven out of sheep stables temporarily by repeated fumigations. It also appears advisable not to pasture sheep on hot summer days in such locations where there are brushes and trees and to exterminate brushes from sheep pastures. Local conditions, however, usually prohibit the carrying out of such measures. It is therefore recommended to apply, in places where sheep flies are common, ill smelling substances to the neighborhood of the nares of the sheep, such as tar, fetid animal oil, hartshorn oil, etc. This procedure, however, is likewise impracticable in the case of large herds. Under these conditions it is advisable, in such neighborhoods where the dangerous flies abound,

to keep the sheep on hot summer days in fumigated barns and to allow them to visit the dangerous pastures only after sunset or at night.

Ruppert observed a decided decrease in the infestation with œstrus larvæ after dipping the sheep at intervals of three weeks in an ordinary scabies dip, and disinfection of their quarters.

Œstrus Disease in Other Domestic Animals. Occasionally the disease occurs in goats. (Hubenet found, in a drove of Bengal goats, many animals which were affected with Œstrus.) The disease is produced by the larvæ of Œstrus ovis and is manifested by symptoms similar to those in sheep.

In **horses** the larvæ of Œstrus (Rhinoestrus) purpureus (Cephalomyia purpurea) infest, in many regions of Europe and in northern Africa, not only the nasal and accessory cavities but also the pharynx and even the larynx. Serious symptoms (cough, dyspnea, laryngeal stenosis) are noted only if the larvæ are present simultaneously in pharynx and larynx.

In **buffaloes** and **dromedaries** the larvæ of Œstrus maculatus (Cephalomyia maculata) are found in similar localizations as in horses.

In **reindeer** Œstrus disease is fairly frequent, and is of importance in so far as it may cause the loss of 1-2% of all animals. During July and August the female of Cephenomyia trompe deposits the larvæ on the muzzle and lips of the feeding reindeer; the larvæ then reach the mouth when the animals lick the muzzle; from there they get into the pharynx, where they cause pharyngitis (see animal parasites in the pharynx).

In nose and pharynx of **deer** the larvæ of Cephalomyia rufibarbis and of Pharyngomyia picta live as parasites. The **elk** is infested by the larvæ of Caphenomyia ulrichii, and the **roe** by those of Cephalomyia stimulator.

In **dogs** Ed. Sergent and E. Sergent (A. P., 1907, 392) have observed in Algeria, in such parts where the number of sheep is small compared with the number of inhabitants, that sheep flies deposit their ova into the eyes, lips and nose of shepherd dogs and of the shepherds themselves, particularly if they eat much sheep's or goat's cheese. The disease called "Thimmi" in the vernacular manifests itself soon after the deposit of the ova, after three to ten days, and during this time there is observed burning of the eyes, disturbance of vision, swelling of the conjunctivæ, lacrimation, also a serous nasal discharge, rubbing of the nose occasionally, symptoms of pain and deglutition, and cough depending upon affection of the pharynx and larynx. In the conjunctivæ and occasionally in vomited material one finds the very small white, lively, motile larvæ. Recovery occurs without exception. The larvæ do not attain full development. Tobacco smoke has proved effective in keeping sheep flies away from men and shepherd dogs.

(b) Pentastomum Taenioides.

Historical. This parasite was discovered in 1763 by Wrisberg; according to Zürn, however, before him by Chabert (1757); its life history was ascertained by Gurly, particularly, however, by Leuckart (1860).

Occurrence. The adult parasite is found preferably among the dogs of butchers, shepherds and hunters; it is found very rarely among other dogs, not at all among pet house dogs. The frequency of occurrence appears to be very variable in different parts of the world.

Among 630 dogs examined in Alfort with reference to this point Colin found the parasite among sixty-four (10.2%); in Toulouse only one (3.3%) out of thirty dogs examined showed the parasite; in Berlin (Deffke) thirteen (6.5%) of 200 dogs; in Chemnitz only 0.92% of 326 dogs examined (Tempel, Feuereissen).

Aside from dogs the parasite has been found in other animals by Chabert, Schwammel, Leblanc, Rose in the nasal cavity of the horse; by Grève, in a mule; Rhind, in sheep; Bruckmueller, in the goat; Bremser, Colin and Miram, in the wolf; and by Laudon, in man. Kulugin found 1 to 2 mature individuals of the parasite in the nasal cavity of calves which five or six weeks previously had taken up ten to sixteen larvae with the food or drinking water.

Fig. 103. Pentastomum taenioides.

Etiology. Pentastomum taenioides, Reed (Linguatula lanceolata [Chabert] s. rhinaria) belongs to the Linguatulidae family of the Arachnoidae (Fig. 103); the body of the parasite is tongue- or lancet-shaped, wider at the anterior end, and gradually tapering at the posterior extremity. The external cuticle consists of about ninety rings and the margins of the body are serrated so that the parasite becomes similar to a tapeworm. The mouth, which opens at the anterior extremity, is surrounded by four hook-like stump feet. The male is 10 to 20 mm.; the female, however, is 8 to 10 cm. long. The ova (Fig. 104) are oval in shape, 90 μ long by 70 μ wide. Special organs of sense, of respiration and circulation are absent.

Evolution. When the eggs are deposited, they already contain embryos and they may be up to half a million in number. They are discharged with the nasal mucus or, if swallowed accidentally, with the feces, the embryos remaining capable of development for several weeks. If the eggs are swallowed by herbivora, with grass or feed, the embryo is liberated in the stomach of the host and passes into the intestine, the walls of which it perforates. With the lymph- and blood-streams it is carried into the mesenteric lymph glands, also into the liver and lungs, less often into the kidneys or the spleen. In these organs the larvæ remain encapsulated for five or six months, undergoing repeated changes of its coverings, and thereby it becomes more like the mature animal. To this stage of development the name of Pentastomum denticulatum (Linguatula serrata) was applied formerly. In the seventh month of evolution, the larvæ leave their capsules and migrate to the serous cavities, also into bronchi and intestines, causing hemorrhages on the way. If they reach the nasal cavity

of a suitable animal, they pass through another process of changing their covering, after which they are mature and copulate.

The infection of carnivora occurs through eating or smelling at organs of herbivora which contain larvæ of Pentastomum: the larvæ may enter the nasal cavities directly through the nostrils or by way of the pharynx, or even from the stomach. In exceptional cases the infection takes place in such a manner that the larvæ are taken up in some way after having accidentally entered the air passages or the intestines and having been discharged with the nasal mucus or with the feces (Gerlach). Chabert, Stiles and Neveu-Lemaire even admit an auto-infection. It is particularly those larvæ which enter the intestines or the bronchi during their wanderings, that invade actively the nasal cavity of the same host.

Anatomical Changes. The mature parasite is usually found in the upper portion of the median nasal ducts and in the cells of the ethmoid bone. It is found exceptionally in the frontal sinuses, in the lower portion of the nose and even in the pharynx (Gellé); the males are particularly liable to get into the larynx when wandering and hunting for the female. The number of parasites varies between one and twelve.

At the site where the parasites are adherent the nasal mucosa is usually reddened, swollen, covered with an abundant secretion or infiltrated with small hemorrhages. Only exceptionally do we find more intense inflammations; occasionally necrosis, especially in the cells of the ethmoid bone. (Concerning the changes produced in the internal organs by the larvæ, see diseases of these organs.)

Symptoms. The Pentastomum taenioides sometimes does not produce any symptoms at all; it causes, however, in the majority of cases, a chronic nasal catarrh accompanied by frequent sneezing (so-called Pentastomum coryza) and subsequent profuse discharge from the nose. The animals rub their noses on the ground or on their front legs or use their paws to rub over it. The nasal discharge is mucopurulent evidently mixed with blood and later on harbors numerous double contoured ova, each of which contains an embryo (Fig. 104). All symptoms are increased after running and sneezing; shaking of the head, rubbing of the nose may appear in the form of paroxysms, during which the animals may expel one or more parasites (Duméril, Mégnin). The olfactory sense is often intensely impaired.

Fig. 104. Eggs of Pentastomum taenioides.

Sometimes there is restlessness and excitement, which is particularly noticeable during sleep, when the animals, which

usually snore, sometimes jump up and sneeze convulsively. While awake restlessness is shown by the fact that the patients are irritable, growl, grind their teeth when approached, hide occasionally, also cry out, turn around in a circle, until they fall exhausted to the floor and lie there motionless. True maniacal attacks, as in rabies, hardly ever occur. Epilepiform seizures are observed rarely.

The difficulties in respiration, which are frequent, may become so severe as to lead to attacks of suffocation, accompanied by whistling and rattling sounds. Exceptionally a perforation of the hard palate may occur.

In **horses** the exceedingly rare nasal pentastomosis is characterized by frequent sneezing, shaking of the head, a mucoid or mucopurulent nasal discharge and chronic swelling of the submaxillary glands.

Course. Nasal pentastomosis always takes a chronic course, because the parasites may remain in the nasal ducts as long as fifteen months (Colin), before being expelled. Most dogs recover from the disease, but they frequently become emaciated. Death is exceedingly rare and is then caused by the extension of an ethmoiditis into the meninges or by suffocation (Dick).

The **diagnosis** can be established beyond doubt only by the discovery of the parasites or their ova. Suspicion of Pentastomum invasion is, however, justified when symptoms of an obstinate chronic catarrh of the nasal and accessory cavities are observed, particularly in butchers', shepherds' or hunters' dogs. The possibility of confounding the disease with rabies appears quite remote. The presence or absence of paralysis is of great differential diagnostic value; the presence of Pentastomum does, of course, not exclude the possibility of rabies.

Treatment. Therapeutic interference becomes necessary only in the presence of severe symptoms. Inhalations of chloroform, dilute ammonia or benzine and instillations of turpentine do not, as a rule, lead to satisfactory results. There is, therefore, nothing left when interference becomes indicated but to trephine the frontal sinuses or the nasal cavities in order to remove the parasites with forceps and to irrigate from above with a solution of creolin or carbolic acid.

Other Animal Parasites in the Nose. In southern countries, **horse leeches** (Limnatis nilotica, Hæmopis sanguisuga), invade not only the mouth and pharynx, but also the nasal cavity of horses, cattle, mules, camels and dogs, causing manifestations of disease by attaching themselves to the mucous membranes. The same is true of the Hemiclepsis (Glossosiphonia) tessellata, a leech which occurs in geese and ducks.

Monostomas belonging to the Trematodes occasionally live in the

nasal and infraorbital cavities of aquatic birds, for instance Monostomum (Cylocoelum) mutabile and Monostomum (C.) arcuatum, without, however, seeming to cause any particular trouble. According to Railliet these parasites, which are found in the infraorbital cavity, have not been identified, and are possibly identical with Hyptiasmus tumidus which was described by Kossack.

Trichosoma aerophilum has been found occasionally in wolves, foxes and, exceptionally, in dogs and cats, inhabiting the trachea and bronchi, although it may accidentally attach itself in the nasal cavity. Ulreich (Vet. 1896, S. 292) found eggs of this parasite in the purulent nasal discharge of a young dog affected with tracheal catarrh. The affection healed in four weeks after the administration of intratracheal injections of oil of turpentine and of sesame. Balla (A. L., 1911, 121) described a case in a pointer that came under observation in the clinic in Budapest. The dog had repeated attacks of epistaxis; in its nasal discharges characteristic eggs of trichosoma were found, and on autopsy the mature worms were discovered in the nasal cavity. The dog came from a region where foxes and wolves are frequent.

(c) Nasal Catarrh in Rabbits Caused by Coccidia. Rhinitis coccidiosa cuniculorum.

(Psorospermiæ, or Gregarinæ Rhinitis [Zürn].)
Coccidiosis oto-rhino-pharyngea.

After the invasion of the nose of a rabbit by coccidia, there occurs an intense inflammation. The affection occurs enzootically and usually spreads to the other mucosæ of the head.

Historical. All enzootic nasal catarrhs in rabbits were formerly known under the collective name of malignant coryza ("malignant snuffles"), but they were all classified as coccidial rhinitis after Zürn had demonstrated the presence of coccidia. Further observations, however, have shown that not all of those affections properly belong to coccidial rhinitis (see page 536).

Fig. 105. Ova of Trichosoma aerophilum in nasal discharge; *a*, ova of trichosoma; *b*, pus cells; *c*, epithelia; *d*, red blood corpuscles.

Etiology. The invasion by coccidia (Eimeria stiedæ s.

Coccidium cuniculi s. C. perforans) probably occurs in the same manner as does the infection by pathogenic bacteria which cause contagious rhinitis (see page 536). Dampness of the bins and the straw appears to be favorable to the appearance and spread of the disease. Young animals preferably are susceptible to the disease; older animals are usually affected slightly and temporarily.

Symptoms. The clinical picture of the disease is very similar to that of contagious rhinitis (see page 537). There is at first moisture around the nares and a scanty mucous secretion. At the same time there is sneezing and sniffing and the animals sometimes rub their noses with their front feet. There is slight fever and accelerated respiration. The sick animals are at first fairly lively and their appetite is good; but after a few days they become listless; they frequently grind their teeth and there is complete lack of appetite. In the meantime, the nasal discharge has become more profuse and the respiration becomes more and more difficult. Frequently, symptoms of conjunctivitis and stomatitis set in. The extension of the inflammation to the middle ear affects the labyrinth and thereby causes the head to be held bent; it also produces staggering and rolling motions. Marked external irritation is apt to give rise to general convulsions.

Later on there is profuse diarrhea, rapid emaciation, and finally death follows.

Diagnosis. A diagnosis can only be established by the microscopic detection of the coccidia which occur in great numbers in the purulent secretion of the nose or of the conjunctival sac. From contagious rhinitis, it can be differentiated by the slight, general disturbances at the onset and the relatively slight rise in body temperature. In the so-called salivation in rabbits, only saliva will be discharged from the mouth in which coccidia can not be found under the microscope. Besides, only small whitish-nodules and vesicles will be found on the lips, while the other mucous membranes of the head are unchanged.

Treatment. Local pathologic conditions may be treated by irrigation or applications of a 3 per cent boracic acid or a ½ to 1 per cent solution of sulphate of zinc or copper sulphate, or of a 1 per cent solution of creolin. Braun recommends in addition the internal administration of flores sulphuris with finely powdered sulphate of sodium in knife-point doses.

Following the recommendation of Raebiger, one can offer medicinal methylene blue, Hoechst (as drinking water in a solution of 1:4000 in boiled water). Furthermore, one may administer daily, atoxyl (0.04 to 0.08 grams) and sodium emeticum (0.066 gm.) in watery solution alternately (Ottolenghi and Pabis).

10. Catarrh of the Maxillary Sinus. Catarrhus antri Highmori.

Inflammation of the mucosa of the antrum of Highmore,

frequently chronic and catarrhal in character, usually leads to the accumulation of a mucopurulent exudate in the cavity of the superior maxillary bone. (Empyema sinus maxillaris sive antri Highmori.)

Occurrence. Catarrh of the antrum of Highmore is observed comparatively frequently in horses, especially in older animals, rarely in other species. The catarrh is usually unilateral.

Among the Prussian army horses during 1899-1908, 0.2% of the total number suffered from this affection, and compared with diseases of the respiratory tract, affections of the antrum were equal to 2% of the former.

Etiology. The disease rarely occurs as a primary affection, as after violent traumatic insults to the maxillary sinus. It is generally secondary to an extending nasal catarrh (see page 523), which spreads to the mucosa of the maxillary sinus which is continuous with that of the middle nasal space through a slit-like opening. Diseases of the teeth and alveoli are likewise frequently the primary cause of the affection, yet often the teeth themselves do not show any affection when inspected through the buccal cavity (Imminger). Caries of the teeth and alveolar inflammations, which are frequently found in horses and occasionally in cattle and dogs, often lead to a spread of the inflammatory process into the mucosa of the maxillary sinus or to an invasion by particles of feed.

In some infectious diseases there may also occur a catarrh of the antrum, as for instance in glanders, malignant catarrhal fever, infectious rhinitis of hogs (see page 532) and of rabbits (see page 536), in coccidia rhinitis (see page 555), in infectious nasal catarrh of birds, in diphtheria of fowls and in fowl-pox. Neoplasms (polypi, sarcoma, carcinoma, cysts) may be causative factors, exceptionally also botryomycosis or echinococcus.

Anatomical Changes. In the beginning of the affection there is noticeable only a more or less uniform or mottled redness and swelling of the mucosa, which later on becomes uneven, nodular, as if studded with millet seeds, and which also exhibits larger gelatinous nodules on sausage-like ridges. If the disease has lasted for a long time, wart-like formations appear on the surface of the indurated mucosa (F. Müller). The cavity of the maxilla contains a discharge which is usually purulent, occasionally fetid, and which may be mixed with particles of food; more rarely the exudate is serons (empyema, hydrops antri highmori sive sinns maxillaris); the walls may be covered with fungi (Nielsen found the mycelium of mucor spinosus). If the opening has become closed the cavity is found completely filled with secretory masses. The anterior wall is thin and protruding. The wall separating the large from the small sinus in the horse may disappear completely.

If the catarrh depends on a primary disease of the teeth, one generally finds, at the inner wall of the cavity, denuded rough bone, or one can feel the passive motility of a tooth through masses of granulation tissue.

Symptoms. **In horses** the symptoms of catarrh of the maxillary sinus follow in some of the cases those of nasal catarrh, and it is the peculiar character of the nasal discharge which arouses suspicion that the antrum is involved. The affection arises, however, frequently without a preceding nasal catarrh, being then usually unilateral in contrast to the cases which develop upon the basis of nasal catarrh and not infrequently are bilateral.

The nasal discharge, which is one of the most frequent symptoms, is at first mucous, soon becomes muco-purulent and later purulent, often lumpy; usually flowing from one nostril only; it may have a foul odor and its quantity is usually very variable. The discharge appears with exacerbations or it may be increased suddenly when the head is lowered, when it is moved in jerks, after blowing or coughing, after rapid motion. It may cease for a time and reappear suddenly, in case the opening which communicates with the antrum has become impassable temporarily by swelling of the mucous membrane or by being clogged up with dried secretions. If the discharge has lasted some time, the resulting maceration of the epidermis leaves an unpigmented groove on the upper lip below the internal angle of the nose. The authors have observed quite frequently lachrymation or even purulent conjunctivitis associated with purulent inflammation of the lachrymal sac owing to the extension of the inflammatory process to the lachrymal duct (Plósz).

In simple catarrh of the antrum of Highmore, a bulging of the wall of the sinus does not occur often and may at first be noticed only on careful inspection of both halves of the face; later, the change in shape may become very noticeable. In case of malignant tumors, on the contrary, the contour of the maxillary sinus is frequently enlarged; extending to the alveoli and to the hard palate, these tumors cause the teeth to loosen, and lead to bulging or even perforation of the hard palate, while the nasal passages often become narrow early; even exophthalmos, strabismus, edema of the lower lid and chemosis may be observed.

On introducing a thin electric bulb or the rhinolaryngoscope into the nose, the affected side will be found less translucent or not at all. Skiagraphic examination of the head shows the collection of fluid by a shadow in the picture of the antrum (which is otherwise clear); the shadow is limited horizontally above and shows undulating movements when the head is moved.

Tenderness of the bony wall of the sinus can be shown to

exist in most cases and is absent only in some of the slight or very chronic cases. In severe affections the infraorbital nerve may be sensitive to pressure.

The wall of the maxillary sinus does not often yield to pressure, but may do so if the bony wall is bulging strongly because of accumulation of secretion or in the presence of malignant tumors. In far advanced cases, fluctuation may be noted.

Dullness on percussion can be shown only rarely, i. e., when the catarrhal secretion or a tumor fills out a portion of the sinus situated above the facial ridge, or if a tumor is connected with the external wall, or if the mucosa is considerably thickened; also if the head of the animal is bent strongly sideways. In case of a partial thinning out of the bony wall of malignant tumors, the percussion sound may on the contrary be louder and deeper, because the thinner bone plate produces more extensive vibrations.

Nasal stenosis occasionally renders respiration difficult, blowing or snoring; it follows upon bulging of the wall of the maxillary sinus together with the inferior turbinate, or may be due to enlargement of a malignant swelling into the nasal cavity.

Enlargement of the submaxillary glands on the affected side is usually present and is acute or chronic, according to the duration of the catarrh. If this lasts for a considerable time, the lymph nodes may become dense, hard, slightly painful, but usually not painful; they may become quite large especially in the presence of malignant tumors, knobby and firmly adherent to the adjacent tissue; in all other cases they remain freely movable.

In **cattle** one usually observes unilateral mucoid or mucopurulent, sometimes bloody, nasal discharges, occasionally mixed with masses of fibrin. The infraorbital region is tender to pressure. Here there may occasionally be dullness and perhaps a protrusion of the wall. A fairly characteristic symptom is frequent blowing with violent movements of the head and the expulsion of mucopurulent masses from the nares. The respiration is snoring and accelerated, but it becomes normal again after the removal of the masses of secretion.

Dogs frequently shake their heads and rub their noses with their front feet. There is also a unilateral seropurulent or mucopurulent nasal discharge, which may be mixed with blood and may be fetid. In the further course the respiration is blowing, due to constriction of the nasal canal. This disappears after opening the buccal cavity or after closing the affected side of the nose. Simultaneously with the appearance of respiratory difficulties, or before these occur, a swelling of the infraorbital region becomes noticeable; it is tender to pressure, hot and later on fluctuating. If fluctuation has occurred, spontaneous rupture takes place with a diminution of the pressure symptoms which may have existed. The opening in the skin

closes within a short time, but a new rupture occurs within one to two weeks and a fistula then persists (Hobday).

In **birds** an inflammation of the infraorbital cavity may develop in the course of diphtheria and of infectious nasal catarrh, exceptionally on account of animal parasites (see page 555);it produces a warm, painful swelling of the region below the eye. On pressure upon this swelling, a mucopurulent mass is discharged from the corresponding nostril.

Course. Catarrh of the maxillary sinus which follows upon acute nasal catarrh or upon a general infectious disease, may develop in the course of a few days and manifest its symptoms in conjunction with those of the primary disease. In other cases the clinical picture develops slowly and may take months before it is complete. Even after the development of the disease the patients remain alive for months and years and are able to work. Later on, however, severe disturbances develop, especially respiratory difficulty, a kind of dumb-staggers, and symptoms of meningitis if the process has invaded the frontal and ethmoidal sinuses.

The involvement of the sphenoidal sinus may lead to disturbances of vision. The exudate may also break through an alveolar space and so lead to the formation of a fistulous tract. Bone necrosis may finally be observed. Especially in horses cases are often mild and may recover spontaneously (F. Müller, authors' observation).

The **diagnosis** is based upon the presence of a unilateral nasal secretion, which is increased in exacerbations through definite causes, of conjunctival catarrh on the same side, unilateral swelling of the glands and the generally existing tenderness of the outer wall of the sinus. A diagnosis can generally be made even if a deformity is not yet present. For the exclusion of glanders, trephining, the mallein test or other immunodiagnostic tests may be necessary. One of the authors' cases, however, proved that a diagnosis, by the aid of trephining, of a non-glanderous catarrh of the sinus cannot always remove the suspicion of nasal glanders. After opening the sinus, it is possible to ascertain whether a simple catarrh is present or whether it is secondary to another primary affection. Very fetid pus creates a suspicion of caries. If the latter is present one can usually detect rough bone on the internal wall of the sinus or a corresponding passively movable molar.—Tumors are palpable and frequently protrude through the wound.—Mucoid degeneration of the bones of the face is distinguished from catarrh of the sinus by a relatively rapid occurrence of respiratory difficulties, marked deformity and softening of the superior maxilla; rachitis and osteomalacia by the fact that the changes are seen on both sides of the face and are not confined to the region of the antrum of Highmore.

Treatment. If catarrh of the antrum of Highmore has developed in connection with rhinitis, deference of treatment appears indicated for one to two weeks, since such cases frequently recover spontaneously. Otherwise trephining of the affected cavity followed by evacuation of the accumulated secretion and local treatment of the inflamed mucosa by repeated irrigation with low pressure leads to recovery. Bad teeth must be removed. A number of disinfectant and astringent solutions may be used for irrigating the cavity; for instance, a 2 to 3 per cent solution of creolin or carbolic acid; 1:1000 corrosive sublimate, 1 per cent solution of alum, tannic acid and sulphate of iron; 0.1 per cent solution of silver nitrate. The irrigations have to be continued until the opening of the antrum is free and until no more pathologic secretion is produced. In birds, incision of the infraorbital swelling, in the longitudinal line of the head, leads to healing provided the primary disease does not take an unfavorable course.

Myxoid Degeneration of the Turbinated Bones and of the Facial Bones in the Horse. Foals sometimes develop myxoid degeneration of the turbinated and neighboring bones. The affection was first observed by Sand; similar cases have since been described by Deupser, Hützen, Nielsen, Fröhner and Johne. Schlegel observed a case of unilateral hydrops of the maxillary sinus, in a heifer, together with mucoid degeneration of the rarefied and extremely light supramaxillary, frontal, lachrymal and nasal bones.

The **cause** of the mucoid degeneration, which develops quite rapidly, is unknown. In the opinion of Merz, the pathological changes, together with the results of histological examinations, indicate ostitis fibrosa which presents many similarities with the local findings in advanced rachitis.

Anatomical Changes. When the prominent region is incised or sawed into, a considerable amount of slimy serous fluid is discharged, mixed with yellow fibrin-like coagula. The antrum is considerably enlarged, the partition walls have disappeared and have been changed into a friable tissue. The internal surface of the cavity is covered with a soft gelatinous material which here and there contains sacs filled with mucus. The turbinated bones may likewise be enlarged and the nasal passages may be closed completely.

Microscopic examination shows that the major part of the bone has been changed into myxoid tissue and that lacunar absorption has taken place. In the neighborhood one sees an active new formation of bone.

The first **symptom** is a nasal discharge which is mucopurulent or purulent, and which becomes more intense after a variable period of time. Simultaneously or somewhat later, especially during exercise, there is difficulty in respiration accompanied by a snoring sound, and these symptoms increase in intensity and become continuous. One observes also early a unilateral protrusion of the facial bones, especially of the superior maxilla; the swelling progresses rapidly and involves either the frontal or the masseter region. The swelling which is at

first of bony hardness later on becomes soft in the region of the greatest prominence, and subsequently in other places; finally there is distinct fluctuation. The laryngeal glands are always enlarged but not painful. Left to itself the affection generally leads to suffocation of the emaciated animals.

The **treatment** consists in timely trepanation followed by disinfectant irrigations. This procedure usually leads to permanent recovery.

11. Catarrh of the Frontal Sinus. Catarrhus sinus frontalis.

Under the name of catarrh of the frontal sinus are designated more or less deeply penetrating, generally chronic, inflammatory processes in the mucosa of the frontal sinuses which lead to the formation of mucopurulent masses of exudate (empyema sinus frontalis).

Occurrence. Catarrh of the frontal sinus occurs only rarely in horses and dogs, more frequently in cattle. Railliet saw one case in a sheep.

Etiology. Traumatic insults causing a hemorrhage into the frontal sinus or a rupture or a fracture of the bony wall of the frontal sinus or a fracture of the horn in cattle, usually play an etiologic rôle. An improper frontal yoke may be the cause of the disease. Joest described a case in a dog in which a plug of cotton was left in the frontal sinus after trepanation, and caused a purulent catarrh. In a certain number of cases catarrh of the frontal sinus developed secondarily as a sequel to catarrh of the maxillary antrum (in horses and possibly also in dogs), or as a sequel to malignant catarrhal fever and finally in consequence of neoplasms in the frontal cavities.

In a heifer, Schlegel saw a case of congenital hydrops of the frontal sinus, with balloon-like bulging and partial rarification of the submaxillary, lachrymal, nasal, frontal and temporal bones.

Symptoms. Corresponding with the usually unilateral affection we often see a unilateral, sometimes fetid nasal discharge, particularly after blowing or after an attack of cough brought on artificially, or after the animal has lowered its head. The discharge is often profuse and is partly licked up by the animals. The first symptom in cattle may be epistaxis, which is later on followed by other symptoms. Firm pressure upon the frontal bone, or percussion of the frontal region or of the base of the horns causes pain; occasionally one may elicit dullness on percussion on either one or both sides. Cattle hold the head obliquely in unilateral affection and lower it in bilateral disease. There may be unilateral or bilateral conjunctivitis with edema of the upper eyelids. In severe cases, particularly in cattle, there may be unconsciousness due either to dull pain in the frontal region (Moussu) or to pressure upon the brain

in consequence of bulging of the internal bone plate into the cranial cavity. In the latter case, exceptionally also under other conditions, one may see the clinical picture of meningitis or of epileptiform attacks. Railliet saw in a sheep the symptoms of gid.

Diagnosis. The disease is easily diagnosed on the basis of the symptoms described, the most important of which are tenderness to pressure of the frontal region and of the base of the horns, elevation of temperature in these regions and nasal discharge.

Treatment. Trepanation of the frontal sinuses is the only proper treatment. Imminger, however, brought about recovery in horses by energetic irrigation by way of the trephined antrum of Highmore.

Moussu proposes that in cattle under three years of age, where the cavities at the base of the horns are not yet well developed and where the lower portion of the frontal sinuses do not much bulge, trepanation should be made in the upper portion of the frontal sinuses in the middle between the median line and the horn pegs. In older animals the core of the horn cavity should be opened 1 to 2 cm. outside of the core of the horn ring and also the lower portion of the frontal sinus, directly over the superciliary ridge, in the middle of the affected half of the front. Irrigation should be performed with lukewarm disinfectant and astringent solutions.
Trepanation in dogs has been described in detail by Weis & Parascandolo.

Catarrh of the Sphenoidal Sinus. The sphenoidal sinus is affected by disease least frequently of all accessory nasal cavities; it communicates with the maxillary sinus by a rather small opening. Yet, catarrhal affections of this accessory nasal cavity are, in all probability, not so very infrequent in horses, and quite recently C. Thomassen [Rev. gén., 1911, XVIII (Lit.)] has called attention to this fact.

Etiology. Catarrh of the sphenoidal sinus is probably always secondary, occurring particularly in connection with catarrh of the maxillary sinus, especially when this contains a large amount of secretion under high pressure, or when irrigating fluids are injected, after trephining, with much pressure. Occasionally general diseases (influenza of horses, purpura, strangles) may give rise to involvement of the sphenoidal sinus even if the maxillary sinus is not affected.

Anatomical Changes. On sawing through the skull, in an imaginary frontal plain between the orbital arch and the maxillary joint, the unilateral or bilateral dilation of the sphenoidal sinus is perceived through a sero-mucous or purulent exudate which may contain cholesterine crystals. According to Thomassen, compression-atrophy of the cerebral nerves, affecting principally the optic nerves and the chiasm, in severe cases the nerves of the eye muscles and the trigeminus occurs often, all these nerves leaving the cranium near the sphenoidal sinus.

Symptoms. Nasal discharge, which is unilateral or bilateral, of similar consistency and character according to the way in which the head is held, as in catarrh of the maxillary sinus, is not always seen in cases in which there is not a simultaneous catarrh of the maxillary sinus. The small opening by which the sphenoidal communicates with

the maxillary sinus, is easily obstructed by slight swelling of the mucous membrane and thereby the outflow of the exudate is prevented; the swelling of the submaxillary glands may also be absent. In consequence the disease is usually manifest only after a time and then in an unfavorable manner through disturbances of the cerebral nerves. There are particularly unilateral and bilateral disturbances of vision (amaurosis) with dilated and fixed pupil, atrophy of the papilla of the optic nerve, congestion and tortuous course of the papillary blood vessels, and at times hemorrhage in the neighborhood of the papilla. Exophthalmos or strabismus may be produced by paralysis of the nerves of the eye muscles. Occasionally manifestations of meningitis may make their appearance.

Treatment. The sphenoidal sinus should be opened from the pharynx and larynx, and the exudate removed. C. Thomassen produces a fissure of the larynx, 15 cm. long, after tracheotomy in narcosis; he anesthetizes the laryngeal mucosa and introduces a trochar of proper length and thickness into the sphenoidal sinus, midway between the openings of the eustachian tubes through the vault of the pharynx; the exudate commences to flow immediately on removing the stilet. In the one case which so far has been operated on in this manner, recovery did not follow because a bit of bone broke off and closed the opening of incision like a valve; moreover the atrophy of the optic nerve had progressed too far.

12. Catarrh of the Guttural Pouch. Catarrhus sacci aerophori.

Catarrh of the guttural pouch is a collective name for acute, or more commonly chronic inflammations of the mucosa of the guttural pouch, which lead to an accumulation of masses of secretion (empyema sacci aerophori).

Etiology. Catarrh of the guttural pouch is due only very exceptionally to traumatic insults (penetration of a ball or a splinter of the fractured lingual bone) or to the penetration of foreign bodies or particles of feed from the pharynx. Whether the moulds found by some authors in the guttural pouch were the primary cause of the disease is still an open question. Catarrh of the guttural pouch is as a rule a secondary affection due to catarrhal or more deeply penetrating inflammatory processes of the deeper portions of the nose or of the pharynx, the process spreading along the eustachian tube which opens during each act of deglutition. Catarrh of the guttural pouch, which occurs in this manner and usually bilaterally, is frequent, according to personal observations and according to examinations made with the endoscope. Exceptionally severe inflammation of the neighboring soft parts (parotid gland, retropharyngeal glands) may stand in an etiologic relation to catarrh of the guttural pouch. The latter affection is only rarely a part of the clinical picture of glanders.

Anatomical Changes. The mucosa is sometimes reddened, also covered with ulcers; if the case has been long standing, it is thickened and uneven; frequently mould colonies are found to have developed. The guttural pouch contains tenacious muco-purulent, occasionally putrid masses in varying amounts, which after desiccation are caseous or more firm (concrementi sacci aerophori) and which have wrongly been called chondroids or gutturoliths. The cavity either contains one large mass of this type or a number of them (up to 200). Owing to decomposition of the secretion there may be a collection of gas (meteorisms sacci aerophori), exceptionally the guttural pouch may be filled with a serous fluid (hydrops sacci areophori).

Symptoms. The clinical picture often develops unnoticeably in connection with severe pharyngitis or rhinitis; in these cases a careful examination of the guttural pouch is suggested by copious nasal discharge when the head is held low, and by the obstinate persistence of the catarrhal symptoms. Less frequently the affection develops gradually without these prodromal symptoms and is then usually unilateral. The clinical picture corresponds in part with that of catarrh of the maxillary sinus.

The nasal discharge is usually muco-purulent or purulent, rather thick or often lumpy. It may appear in exacerbations or it is increased in amount when the head is held low, when it is shaken, or during motion; it ceases after the exudate has become desiccated in the guttural pouch.

Swelling of the submaxillary glands is not often absent and may vary in degree; it increases gradually and may be acute or chronic; the lymph nodes do not become adherent to the surrounding tissue. In acute cases they may suppurate.

The swelling of the region of the parotid gland (Fig.106) varies in degree, according to the expansion of the guttural pouch by the accumulated exudate; in some cases it may be recognized only by careful examination of both sides in the region of the throat; in other cases it may be sufficiently marked to obliterate the depression between lower jaw and atlas, or the entire parotid region may bulge out and the swelling may, in exceptional cases, extend to the middle third of the neck between trachea and vertebrae (Fig. 106). The swelling is frequently unilateral and in coexisting pharyngeal catarrh it is difficult to differentiate it from the swelling of the region of the gullet. There is little or no tenderness on pressure, the consistency is soft or fluctuating, sometimes tense and elastic; frequently pressure causes it to diminish in which case the nasal discharge becomes more copious from the nostril on the same side, or it may make its appearance only then. A splashing sound is heard sometimes, when the animal moves the head suddenly or if palpation is practiced by thrusts.

The percussion sound is dull over the entire swelling or only in its lower portions; if much gas has formed, the sound has a loud *tympanitic character, particularly in the upper portions.* In the latter case a metallic sound or the sound of the *cracked pot may be noted.*

In severe cases, rhino-laryngoscopic exploration from the pharynx discloses beside and in front of the arytenoid cartilage, an indentation of the lateral pharyngeal wall, of variable size, toward the pharynx which, in consequence, becomes more narrow, and the epiglottis is covered more or less (*Fig. 107*). Stringy mucopurulent exudate often comes from the opening of the eustachian tube (Fig. 10 especially if a vigorous pressure is exerted, in dorso-medial direction, upon the lower portion of the parotid region on the same side.

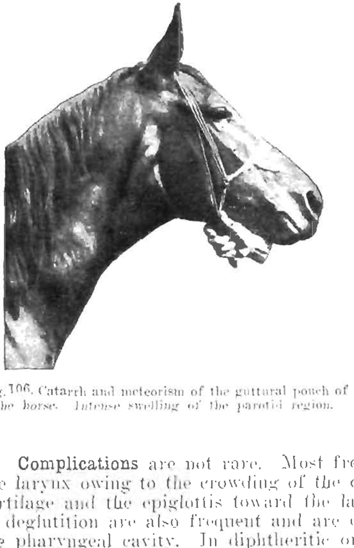


Fig. 106. Catarrh and meteorism of the guttural pouch of the horse. Intense swelling of the parotid region.

On direct endoscopic examination of the guttural pouch with a thin endoscope, the accumulations of pus can be seen on the bottom of the cavity (Fig. 109), or the end-piece of the endoscope, to which the lamp is attached, enters the exudate, in which case the entire field assumes a uniform yellowish-white color. In this manner air-sac catarrh, usually bilateral, may often be recognized in the course of pharyngitis, even if its external signs are entirely obscured by those of the pharyngitis.

On fluoroscopic examination, the lower portion of the air-sac picture, which normally is very light, is found to be darkened more or less, being limited horizontally; movements of the head cause wave-live motions.

Complications are not rare. Most frequent is stenosis of the larynx owing to the crowding of the equilateral arytenoid cartilage and the epiglottis toward the larynx. Disturbances of deglutition are also frequent and are due to narrowing of the pharyngeal cavity. In diphtheritic or necrotic inflammations attacks of nosebleed may take place, and even fatal hemorrhage after destruction, by ulceration, of the large blood vessels which are near the air-sac cavity (Art. carotis externa and interna; Art. maxillaris externa and interna); the blood is then discharged through the opening of the eustachian tube into the nasal cavity and outwards (Plósz, Enger, Wester). Another complication, which is not infrequent, is aspiration pneumonia owing to the disturbances in deglutition, which cause the guttural pouch to be filled with coagulated blood and ma

simulate paralysis of the gullet (Wester). A case observed by Zschokke shows that paralysis of the gullet may actually take

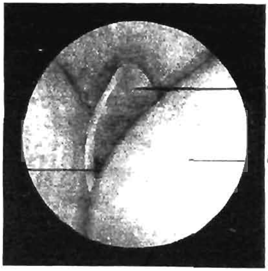


Fig. 107. Extension of the guttural pouch into the pharyngeal cavity and partial obstruction of laryngeal opening: **a**, epiglottis; **b**, tympanic extended left guttural pouch; **c**, laryngeal opening.

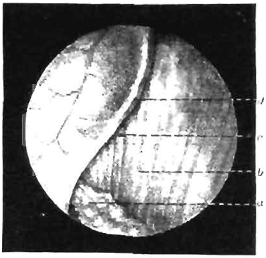


Fig. 108. Purulent discharge from the orifice of the left eustachian tube: **a**, pus; **b**, lateral pharyngeal wall; **c**, orifice of extension tube; **d**, its median wall.

place if the inflammation is extended to the near-by nerve trunks. In a case reported by Durante, perforation of the eardrum was followed by a mucopurulent discharge from the ear; this discharge could also be made to flow by vigorous pressure upon the diseased guttural pouch. Occasionally the catarrh is followed by *purpura*.

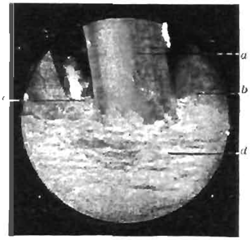


Fig. 109. Endoscopic view of the right guttural pouch with empyema, **a**, large branch of the thyroid bone with marked swelling of the mucous membrane; **b**, median; **c**, lateral, **d**, anterior part of the guttural pouch filled with a purulent exudate.

Treatment. If catarrh of the guttural pouch is acute and makes its appearance in the course of pharyngitis or rhinitis, deferred treatment is indicated for a period of 1 to 2 weeks, because in these cases the catarrh often heals spontaneously, or at least the exudate dries up and causes no further trouble. *Recent cases may be cured by* opening the guttural pouch, cleaning it out and irrigating repeatedly. Any concrements that are found are removed with forceps (Woodward). If, however, the enlargement of the guttural pouch is already quite considerable, and if the sac has become adherent to neighboring structures, the prognosis as to recovery is very bad. Irrigation

of the guttural pouch with a Guenther catheter is not advisable on account of the danger of aspiration.

Bloating of the Guttural Pouch. (Tympanitis s. meteorismus sacci aerophori.) Bloating of the guttural pouch (meteorismus idiopathicus) is sometimes seen in foals as a primary affection. The cause of it is probably paralysis of the muscles of the eustachian tube, in consequence of which active dilatation ceases, which causes the expulsion of air (Gerlach, Dieckerhoff, Peter). Exceptionally bloating of the guttural pouch may be due to congenital anomalies (the presence of folds of mucosa which close like valves [Niebuhr, Thomassen]). The affection occurs secondarily in consequence of chronic catarrh of the guttural pouch (*meteorismus symptomaticus*).

The **symptoms** are a puffy, elastic swelling in the region of the parotid (Fig. 106) which may be unilateral or bilateral; on percussion a tympanitic sound is elicited; the swelling may decrease upon pressure and a peculiar noise may then become audible. Cases are also observed in which the swelling is only found in the pharynx. Difficulty in respiration is always observed, difficulty in deglutition occasionally. In very serious cases pressure upon the bloated guttural pouch produces whistling or roaring sound during respiration. Nasal discharge or swelling of lymphatics are absent in idiopathic bloating of the guttural *pouch*.

The **treatment** consists in opening the guttural pouch, if necessary also in artificial dilatation of the tubal opening, and irrigation.

SECTION II.

DISEASES OF THE LARYNX.

1. Catarrh of the Larynx. Catarrhus laryngis.

(Laryngitis catarrhalis, Angina.)

Catarrh of the larynx develops, at least in its acute form, as a superficial inflammation of the laryngeal mucosa in connection with catarrh of the trachea (tracheal-laryngeal catarrh); also in connection with catarrh of the pharynx (catarrh of the upper respiratory passages). These affections may be considered under the head of laryngitis because the symptoms of the latter stand in the foreground of the clinical picture.

Etiology. One of the most common causes of acute laryngeal catarrh is taking cold, hence the disease is frequently seen in spring and fall. Laryngitis is also frequently due to the inhalation of impure dust, or of otherwise irritating air. Alcohol contained in distillery mash probably has a similar effect, and cattle fattened on such mash frequently develop the symptoms of acute laryngitis (so-called distillery mash cough).

Irritating and traumatic insults may likewise produce a noxious effect, as for instance in sudden and awkward drenching with fluid medicines or in the introduction of pills with a stick or the removal of foreign bodies wedged in the pharynx of cattle. The larynx is also irritated mechanically by long-continued convulsive cough or barking or bellowing.

A laryngitis may develop secondarily by the continuation of an inflammatory process of the nose, the pharynx, the trachea, the bronchi and of the lungs (catarrh, lungworm disease, pneumonia, etc.).

It is of importance that the secretions from the pharynx and from the deeper respiratory tract may come in contact with the larynx. Infection is frequently the cause of laryngitis in a number of infectious diseases (strangles, influenza, distemper, malignant catarrhal fever, tuberculosis, glanders, etc.).

Chronic laryngitis is produced by similar causes as those which are responsible for acute laryngitis, if these causes act

upon the mucosa for a longer time or repeatedly. Consequently chronic laryngitis is observed particularly among such animals as are kept permanently under unfavorable conditions, especially in impure air. In old dogs one frequently sees an obstinate chronic laryngitis.

Other causes are: disturbances of circulation in chronic heart disease, chronic inflammatory diseases of the deeper respiratory passages and of the neighboring organs, new formations (cysts, fibroma, sarcoma, carcinoma, or other tumor-like swellings—polypi, actinomycosis, tuberculosis, glanders).

Susceptibility. Idiopathic laryngitis is seen particularly in pet animals with little power of resistance, for instance in horses kept in warm stables and in house dogs.

Anatomical Changes. In acute laryngitis the epiglottis, the arytenoid cartilages and the vocal cords are intensely reddened and swollen; in a later stage they are abundantly covered with mucus and sometimes infiltrated with blood. In severe cases there occur round superficial ulcers (Bruckmüller).

In chronic catarrh the swollen parts are more grayish in color; in consequence of the swelling of the mucous glands or proliferation of the epithelial cells the surface is finely granular (laryngitis granulosa). The mucosa is thick and may in obstinate cases be set with villous excrescences; in horses retention cysts may form.

Symptoms. The principal and most common symptom of acute laryngitis is cough; it is at first short, dry and rough, later drawn and moist, always, however, more or less painful. In the presence of great tenderness it may be weaker. The animals are at short intervals subject to a tormenting cough, sometimes in the form of convulsive attacks. Dogs sometimes vomit after coughing. These attacks generally come on after the action of an external irritant on the larynx, especially after the animal has been led out of the barn, after a draft of cold air through the barn door, after the ingestion of cold water, or of dusty or hot feed, or after sudden or prolonged exercise. In rare cases a scant nasal discharge is noted, which is serous, mucous or muco-purulent. The submaxillary glands may show moderately acute swelling.

Owing to the increased sensitiveness of the laryngeal mucosa, the animals try to escape pressure on the larynx; even slight pressure gives rise to repeated coughing. In more severe cases the head is stretched out moderately.

Difficulty of respiration exists only when the mucosa is swollen more markedly. In such cases one also hears whistling and rattling noises during inspiration. These symptoms may finally lead to the clinical picture of edema of the larynx. One also hears frequently rattling over the larynx and the windpipe. In horses and dogs one may observe hoarseness.

Fever exists only in the initial stage of simple laryngeal catarrh. The elevation of temperature, however, as a rule, amounts only to a few decimals of one degree (C.°). Catarrh of the larynx occurring during the course of infectious diseases is associated with moderate or even high fever.

Chronic laryngeal catarrh. The symptoms are on the whole similar to those of the acute form. As a rule, however, the cough is less painful, rough, crowing and dry; rarely somewhat moist and accompanied by a peculiar explosive sound. The inspiration following cough is occasionally characterized by an audible noise. The secretions which collect during the night cause long-continued attacks of cough in the morning. The sensibility of the larynx is often increased only slightly. Usually the respiration is not disturbed; in exceptional cases stenosis of the larynx develops on account of proliferation of the mucous membranes or of new-growths.

Course and Prognosis. Primary acute catarrh of the larynx lasts from a few days to one or two weeks. The disease rarely persists for weeks and months. If the duration is very long the mucosa undergoes changes which exclude the possibility of complete recovery. Acute exacerbations are not rare in chronic catarrh.

Treatment. The most important measures to insure a speedy recovery are rest and protection against external irritation. The patients ought to be placed in a moderately warm place, with pure, quiet air. They ought to receive dust-free feed; if possible, green mashed food and bulbs. When the weather is favorable the open air is particularly beneficial in chronic catarrh. Animals which are only slightly sick may be used for light work.

If a tormenting cough is frequent the tenderness of the larynx should be lowered by narcotics. Indicated are: morphium (℞ morph. mur. 0.10, aq. amygd. amar. 12.0 M. S. every 2 to 3 hours, ten to fifteen drops for dogs); also codeine (℞ codein. phosphorici 0.60, aq. dest. 150.0 M. S. every 3 hours, 1 to 2 teaspoonfuls for dogs; heroine (℞ heroini mur. 0.10, aq. dest. 150.0 M. f. sol. S. every six hours, 1 to 2 teaspoonfuls for dogs). Larger domestic animals should receive these drugs in the proper doses by subcutaneous injection; heroin causes great excitement in horses and occasionally a strong depression in dogs. The salts of bromine alone or in combination with codeine are sometimes indicated.

Priessnitz' compresses to the region of the larynx (every 2 to 4 hours, in dogs every 1 to 1½ hours [do not leave over night]) are very serviceable in case they are tolerated by small animals. In case of dogs with a very tender skin, dry compresses should be used instead (particularly in chronic ca-

tarrh), or none at all. Warm packs are less effective. The inhalation of water vapors is of good effect in the incipient stage when the mucosa is comparatively dry and very irritable. Tenacious mucus may be made more liquid by the inhalation of finely divided muco-solvent remedies (1 to 3 per cent solution of common salt, sodium carbonate or bicarbonate). In the presence of an abundant thin fluid secretion, astringent and disinfecting drugs are indicated, such as alum (1-30%), chloride or iron (0.1-0.3%), creosote (0.5-1.0%), nitrate of silver (0.1-0.5%), oil of turpentine, tar (aqua picis 10%).

Inhalations are applied in the same manner as in nasal catarrh (see page 528). The inhalation of aqueous vapors or fluids which easily evaporate does not require any special apparatus, while the spraying of other fluids can only be accomplished by special devices. Ordinary sprays are not very serviceable; more to be recommended are Siegel's or Bullin's inhalers, the best is Wasmuth's apparatus, which, however, is generally only available in veterinary clinics, on account of its high price. Horses and cattle must be made to inhale through the mouth, which if necessary must be kept open by a speculum. Inhalations ought to be repeated every three or four hours and should be of ten tn fifteen minutes duration. In the treatment of smaller animals one may employ tubs with boiling water covered by a perforated board; the patients are placed on the latter. Birds are held over a vessel containing hot water or over an inhalation apparatus, their mouths being held open. For the treatment of cage birds, a vessel filled with warm water may be placed beside the cage, and the whole covered with a cloth, or the spray of the solution is directed into the cage, but in this case care must be taken that the bird can breathe a sufficient amount of air.

Only a very small fraction of a spray finds its way into the larynx of larger animals; hence, when treating them a Frick or Bayer-Kieselbach spray apparatus should be employed. These are easily introduced in horses; in cattle they can be introduced with some difficulty into the pharynx like a rhinolaryngoscope, and in this way permit of a more direct spraying of the larynx. If convulsive cough is present the solutions ought also to contain morphine up to ½% in amount.

The best results are to be expected in chronic laryngitis from painting the mucosa of the larynx or from the insufflation of powders, in cases in which the animals do not get too much excited during such manipulations. Indicated are: acidum tannicum or alum, argentum nitricum and saccharum album āā; cocaine, eucain, orthoform (of each 0.01 gm. with 0.2-0.3 sugar). The painting is best done with a 1-5% solution of silver nitrate.

The powder is applied to smaller animals either with a long and properly curved brush or with a feather or by the aid of a Rauchfuss powder-blower. Painting may be done with a brush or with a sponge fastened to a small rod. The applications must be repeated daily.

In larger animals one may use a Frick or a Bayer spraying apparatus, and powder may be applied with a Neubarth powder-blower.

Intratracheal injection (Dieckerhoff) may be performed in larger, as well as in smaller, animals. Larger animals receive 30 to 40 cc.; smaller animals 4 to 5 cc. of fluid, which is rapidly injected with a needle immediately behind the larynx in a direction toward the pharynx. The following solutions are used: 0.5% solutions of alum, tannic acid or sulphate of zinc;

0.3% solution of acetate of lead; 0.1% solution of silver nitrate; in large animals if the cough is very tormenting morph. mur. is indicated in a 1% solution (10-20 or 0.5 to 2.5 gm.). Although intratracheal injections are in general held to be without danger, they can usually be dispensed with, because they require a considerable dexterity in dogs and hogs, as well as in other animals.

If the laryngitis is contagious in character, isolation of the healthy, or at least, of the sick animals, and disinfection of the stable or barn is indicated.

Epizootic Laryngo-tracheitis of Horses. A usually very contagious catarrh of the respiratory passages of horses has been known since 1888. It usually affects the mucosa of the larynx and trachea; at other times that of the bronchi, or again that of the respiratory parts situated anteriorly to the larynx. According to its variable location the affection is known as iufectious laryngitis, infectious enzootic catarrh of the upper respiratory passages, enzootic cough, laryngo-trachéite épizootique (French), bronchite infectieuse, also frequently "la grippe." To this group probably also belongs the contagious pharyngitis of Dieckerhoff. According to Meyer "Skalma" (Dieckerhoff) is also a bronchial complicatiou of this affection.

Etiology. The disease has much in common with equine influenza. Finzi has recently shown experimentally that it is etiologically identical with equine influenza and that it represents only a special form of this disease. Healthy horses contracted typical cases following intravenous injection of filtered blood from sick horses. It is usually observed annually. The extent of its prevalence, however, varies considerably in different years. Its very contagious nature is responsible for a frequently rapid extension within a few days among the horses of the same stable so that it assumes an enzootic and not uncommonly an epizootic character. The introduction of the disease occurs with newly acquired horses, with remounts, etc. The contagion may also be conveyed by persons or by the air. Younger horses are most susceptible. In some epizootics, however, horses are affected without reference to age. The period of incubation is 1 to 5 days according to past experiences.

Symptoms. The most prominent symptom, which is never absent, is a dry, short, strong, rough cough. If it is painful at all it is only moderately so. The cough occurs either very frequently or on the contrary after longer intervals. A marked tenderness of the larynx and of the trachea can be demonstrated. A serous, later on mucous, but usually scanty nasal discharge comes on which subsequently often becomes more abundant and lumpy. If this is the case the lymph glands of the laryngeal entrance swell moderately and mucous râles are heard over the trachea. In some cases bronchitic symptoms become manifest. The conjunctivae often appear normal or if there is an elevation of temperature they are markedly reddened, often diffusely rose red or yellowish-red or even dark red; sometimes, on the contrary, pale; according to Meyer edema is seen in the lower portions of the legs, the abdomen and the sheath of the penis.

Fever is often present. The temperature usually does not rise above 39° C., but it may not uncommonly go up to 41°. Even if

occasionally fever is not present at rest, more intense exercise will elevate the temperature and at the same time accelerate the respiration and the pulse beat. Many patients show lassitude and malaise which contrasts with a comparatively insignificant psychic depression.

Concerning weakness or paralysis of the hind extremities (lumbar cord disease) following catarrh of the upper respiratory passages as observed by Kull and Duvinage, see enzootic paralysis of the cord.

The **course** of the disease is generally benign; the disturbances disappear in 8 to 10 days and after 8 or 10 days more the animals are able to work again. In some epidemics and in foals under two years old the period of convalescence lasts several, up to 6 to 8 weeks. Sometimes pneumonia or pleuritis comes on as a complication, especially if the animals have been worked hard during the disease. One attack of the disease does not appear to insure any lasting immunity.

The **treatment** consists more properly in feeding mashes, beets or a dust-free dry feed, sojourn in the open air during the favorable season, even light work in the open. These measures are better than the administration of medicines. Following treatment with salvarsan, Barthel observed a rapid disappearance of fever and pronounced improvement in the general condition. Sometimes change of location brings about a disappearance of the epizootic. Complications which may come on require proper special treatment. The stables must be thoroughly disinfected.

Epizootic Laryngo-tracheitis of Cattle. In 1890 there was observed in some parts of Bavaria an acute catarrh of the respiratory passages in cattle which spread enzootically and even epizootically, and which was believed to stand in some relation to an influenza epidemic which was then very prevalent in man. More or less similar enzootics and epizootics have since been observed repeatedly, and they have been designated as epizootic laryngo-tracheitis (Zimmermann) or as influenza-like affections (Bräuer, Prietsch), or as infectious catarrh of the respiratory passages (Schmidt, Lewek). According to Lewek all these various affections are one and the same infectious disease, peculiar to cattle, which makes its appearance either sporadically or enzootically, particularly in south Germany. Cattle distemper (Janson) or influenza of cattle (Harms) deviates materially from the above affection, but they may be here considered, since their true etiology has not yet been cleared up.

Epizootic laryngo-tracheitis or the influenza-like affections of cattle are contagious and are often imported by newly acquired animals (Zimmermann, Schmidt). The disease is evidently of bacterial origin. (In 8 cases examined Lewek found Gram-positive bacilli four times, once Gram-positive cocci and three times a mixture of both.) A longer transportation on railroads or aboard of ships seems to predispose to the infection and the disease is therefore often observed in cattle after shipment. According to Zimmermann the period of incubation is two to three, and according to Lewek, one to four days.

A contagious catarrh of the upper respiratory passages of epizootic character has also been observed in goats

Symptoms. We first observe a dry, painful cough coming on in attacks. The temperature rises to above 41° C., there is marked tenderness of the larynx and of the trachea, also marked reddening of the nasal mucosa and in the further course a watery or mucous nasal

discharge associated with a mild conjunctivitis, occasionally also swelling of the eyelids. Rough, whistling, later on drawn and rattling, noises are heard over the region of the larynx. If bronchitis is present there is difficulty of respiration; the latter may also be due to a broncho-pneumonia which develops after two to three days in 50% of the cases (Schmidt). The appetite is usually much diminished. The disease usually lasts one to two, more rarely, two to three weeks, and ends, as a rule, in recovery. A fatal termination may occur in cases complicated with broncho-pneumonia.

In influenza-like affections of cattle one also observes the symptoms of gastric catarrh, arthritis and mastitis, to which may be added, in some cases, the signs of inflammation of the deeper structures of the eyes and a marked depression and lassitude on motion. Even in these cases, however, the course is usually benign.

Treatment is only indicated in those cases in which complications occur.

Japp Disease of Lambs. Under this name Hasenkamp (D. t. W., 1909, 300) described a disease of lambs observed frequently in the provinces of Hanover and Westphalia in Germany. This disease only occurs while the animals are stabled and is characterized by an intense reddening and swelling of the mucosa of the larynx and trachea; by albumoid degeneration of the muscles of the larynx and by congestion of the lungs. Bacteriologic examination of the internal organs of animals dead from the disease has been negative. The affection has generally been looked on as mercurial poisoning after treatment of sheep-scab with a mixture of therosot. This view, however, is contested by Hasenkamp.

Whooping-cough (Pertussis). In human medicine, the term whooping-cough is employed for an infectious disease which is localized in the respiratory passages and usually affects only children. According to Bordet & Gengou, it is due to a small, ovoid, Gram-negative bacterium and is transmissible to young animals by means of its cultures. An incubation-period of 3 to 14 days is followed by an acute catarrh of the upper air passages, to which the characteristic paroxysmal cough is added one or two weeks later. The paroxysms occur suddenly and consist of a rapid succession of short, convulsive coughs, until the entire inspired air has been expelled; then a deep inspiration follows, which is accompanied by a whistling sound, and after this again a series of cough-spasms. The attack terminates with gagging or vomiting; it usually recurs at short intervals and may be induced by definite causes (fright, crying, depression of the tongue). The disease lasts from 4 to 8 or 12 weeks.

Markert (M. t. W., 1910, 385) observed in young dogs and their bitch, a disease with symptoms very similar to those just described, in a place where whooping-cough was prevalent among the children; it can therefore not be denied that the disease was transmitted to the dogs, and it is quite possible that dogs may acquire true pertussis. The paroxysms persisted for about 4 weeks after the dogs had been removed to the country, and then disappeared without leaving a trace.

2. Croupous Laryngitis. Laryngitis crouposa.

(*Häutige Bräune, Halsbräune* [German]; *Laryngitis fibrinosa, angina membranacea, croup.*)

Croupous laryngitis is characterized anatomically by fibrinous membranous deposits on the laryngeal mucosa and is frequently associated with a similar affection of the trachea, occasionally also of the pharynx.

Occurrence. Croupous laryngitis is generally a rare disease. It is most commonly seen, if met with at all, among

cattle and sheep; more rarely among horses, pigs and cats. It occurs occasionally in enzootic distribution. Guittard once saw an enzootic of croupous laryngitis among geese.

Etiology. Croupous laryngitis is primarily caused by the inhalation of hot air, smoke and other irritating substances (ammonia, chlorine gas, sulphurous acid, etc.). Cases which are occasionally observed after conflagrations or after disinfection of stables with irritating vapors are of such origin.

According to Roche-Lubin, the disease is also caused in some places by the practice of chasing sheep around for twenty-four hours in dusty places in order to increase the weight of the wool.

Aside from affections of the above nature, there is seen in cattle, sheep, horses, pigs, cats and young geese, a croupous laryngitis due to an infection. Catching cold appears to play a predisposing rôle, because most of these cases are seen in spring and fall, on damp days or after sudden changes of temperature, also after a sojourn in the open during cold nights (according to Forneris often seen in the mountains of Piedmont). The microbe causing this infection is not as yet definitely known. Some observations, however, of Ernst seem to point to the bacillus necrophorus as the etiologic factor. This bacillus is frequently present upon the mucosae of herbivora and it may do harm if the resistance of a mucosa is lessened or if the bacterium has attained a higher degree of virulence. Other bacteria, perhaps streptococci, may have a similar effect.

The disease sometimes occurs secondarily in infectious diseases with similar or diphtheritic inflammations of the neighboring organs. Such diseases are: malignant catarrhal fever and croup of cattle, diphtheria of fowls and calves, rinderpest, sheep-pox, purpura hemorrhagica of horses, acute glanders, etc.

Anatomical Changes. Elongated, cylindrical or tubular fibrinous pseudomembranes are found at the base of the epiglottis, at the epiglottic-arytenoid bands, and also at the other portions of the larynx, and not uncommonly farther down in the trachea and even in the first portions of the bronchi. The membranes are 1-3 mm. thick in smaller animals; in larger animals considerably thicker grayish-white, or brownish in color. They are sometimes quite loose, almost liquid; at other times firmer and dryer. They are frequently partly detached, especially at the periphery, and still adherent in other portions. Under the pseudomembranes the mucosa is intensely reddened, sometimes containing extravasated blood, and in the deeper layers infiltrated serously or purulently. Sometimes the inflammation shows a diphtheritic character and deep losses of substance are then seen after the removal of the pseudomembranes.

Occasionally mirasmatic ulcers of the larynx are found, due to pressure in extremely emaciated animals, and in the course of purpura hemorrhagica in the horse (Joest), and also in very sick animals which, prior to death, show difficult respiration for days (author's observations). Such ulcers are usually located symmetrically in the dorsal region of the larynx on the internal aspect of both arytenoid cartilages or at the lower extremity of the vocal cords—that is, in places that come in contact during respiration. The base of the ulcers is covered with yellowish-gray smeary masses, the edges are rather sharp and smooth, and the mucosa slightly reddened. Fayet, Leysses and Prudhomme found in epizootic lymphangitis an ulcer in the form of a granular loss of substance with a purulent deposit on the posterior half of the vocal cord at the base of the arytenoid cartilage.

As sequelae of this affection may be mentioned purulent bronchitis, catarrhal or croupous pneumonia, atelectasis of the lungs, acute swelling of the cervical and intrathoracic lymph glands in cattle, also frequently croupous gastritis and enteritis.

Symptoms. Croupous inflammation of the larynx begins with the symptoms of an acute laryngitis which increases rapidly in intensity; the respiratory difficulty reaches a high degree within 1 to 2 days.

The temperature is considerably elevated from the start. Cough comes on soon; it is at first dry, short and strong; after a short time, however, it becomes peculiarly hoarse, convulsive and very painful. The respiration assumes a character pointing to stenosis of the larynx sometimes during the first day; the inspiration is markedly forced. Each inspiratory act is accompanied by a peculiar whistling and rattling sound. Over the regon of the larynx and the trachea stenosis sounds can generally be heard associated with moist râles and the hand placed over the larynx feels distinctly a laryngeal fremitus. Increased laryngeal tenderness is permanently present, and even mild pressure brings about a convulsive cough, and even an attack of suffocation.

During the attacks of cough the animals expel an abundant mucopurulent or purely purulent secretion through the nose and mouth. Toward the third to fifth day membranous shreds of fibrin are thrown out in addition or also longer fibrin cylinders, the caliber of which agrees with that of the larynx and trachea. The difficulties in respiration cease markedly directly after the expulsion of such masses and the general condition of the patient improves rapidly and may go on to complete recovery. In other cases the improvement is only temporary or amelioration and aggravation alternate repeatedly.

The neighboring lymph glands are swollen and tender; the pulse is rapid, small, and later on almost filiform. The appetite is poor; the feces are sometimes mucoid or mixed with shreds of fibrin. The secretion of milk ceases from the start.

Course. In a part of the cases death occurs on the third and, in young geese, even during the first two days from suffocation. Except in the last mentioned birds, the course is generally somewhat slower; does not, however, as a rule, extend over more than ten days, and is unfavorable in most cases.

Diagnosis. Croup of the larynx can only be diagnosticated definitely when the animals cough up shreds of pseudomembranes or void them during an attack of vomiting, or if the membranes can be seen directly in the larynx of the smaller animals. The laryngeal stenosis, however, which develops rapidly within one to two days with fever and in connection with a peculiar hoarse cough, usually furnishes enough data to arrive at a probable diagnosis. In consequence of simple catarrh such marked stenosis does not develop while after the lodgment of foreign bodies or of animal parasites (Östrus, Pentastomum), or in acute edema of the glottis a high degree of stenosis is manifest within a few quarters of an hour. In stenosis due to neoplasm, there are no symptoms of an acute affection.

Prognosis. In small or younger animals the prognosis is very unfavorable, and recovery is the exception even in large and adult animals. In making a prognosis the general conditions of the animals should be considered; weak and otherwise diseased animals will hardly ever get over an attack. The expulsion of abundant masses of fibrin is a favorable symptom. The involvement of the lungs excludes recovery absolutely.

Treatment. The expulsion of the croupous membranes may be hastened in smaller animals by the use of emetics (sulphate of copper 0.05-0.5 gm., ipecacuanha 0.5-2.0 gm., tartar emetic, 0.1-0.3 gm.); if difficulties in deglutition exist these drugs are applied in the form of clysmata, or apomorphia may be given hypodermically (dogs 0.003-0.01 gm., cat 0.002-0.005 gm.) in aqueous solution. Hogs receive veratrine (0.02-0.03 gm. diluted in alcohol), likewise hypodermically. Vomiting must particularly be brought about in the presence of a sudden attack of suffocation, which is generally due to the lodgment of fragments of pseudomembranes in the rima glottidis. Detachment of the membranes is also favored by repeated inhalations, every one to two hours, of finely divided alkalies; lime water is principally used. If suffocation threatens tracheotomy must be performed. This operation may save the life of the animal whenever the inflammatory process is confined to the larynx or to the larynx and trachea, while the lungs are not affected. One may also try inunctions with mercurial ointment in the region of the larynx, moist warm or warm fomentations, excitantia (alcohol, wine) and the patients must sometimes be nourished artificially.

Breton and Larieux saw good results following the use of antidiphtheria serum. Instead of this, according to the experiments of Schmiedhoffer, probably any foreign normal serum could also be injected with success.

3. Edema of the Larynx. Oedema glottidis.

By edema of the larynx, or oedema glottidis, is understood an accumulation of serous fluid in the loose submucous connective tissue of the epiglottic-arytenoid folds and of the ventricles of Morgagni, in consequence of which stenosis of the larynx is brought about.

Etiology. Edema of the larynx occurs as a primary affection after the inhalation of hot air, irritating acrid gases or dust-like substances; also after injury of the laryngeal mucosa; its development may be favored by preceding hard work, especially by excitement which has led to hyperemia. In this manner one may explain the occurrence of edema of the larynx after forced marches on dusty country roads (Buguiet) and at conflagrations. In young ducks fatal edema of the larynx is sometimes caused by the sting of bees which have fallen into the water when they were tired out and have been ingested by the young ducks.

In the overwhelming majority of cases, edema of the larynx is a secondary affection, coming on principally during severe disease of the larynx or of the organs in the immediate neighborhood of the former (pharynx, tongue, parotid gland, laryngeal and retropharyngeal lymph glands). As a collateral edema the affection is seen in certain infectious diseases (anthrax, hemorrhagic septicaemia of deers, of buffaloes, hogs and in hog cholera, petechial fever, pox, etc.); urticaria in cattle is sometimes associated with œdema glottidis (Wyssmann, Albrecht). Edema of the glottis develops not infrequently in the course of serum-disease.

Congestion (chronic heart disease), compression of the jugular vein, traumatic pericarditis (Liebetanz) or cachexia may give rise to edema of the glottis; such cases are however rare when compared with various edemas in other places of the body.

The fat collecting in large amounts around the entrance to the larynx in fattened hogs produces, according to L. Weisz, an enlargement of the thyroid gland by compression of the veins, and the pressure of the thyroid upon the larynx causes a whistling sound (animals so affected are called "lung-whistlers.")

Anatomical Changes. The epiglottic-arytenoid folds, the lateral bands of the vocal cords, the folds between the epiglottis and the tongue, form trembling gelatinous ridges with either a reddened or a pale surface. These ridges collapse after an incision, and discharge a clear, more rarely turbid, serous fluid, and the mucosa then forms folds. Exceptionally the connective tissue of the ridges is found infiltrated with a purulent exudate, especially if the edema occurs in connection with deeper in-

lammation of neighboring parts (perichondritis, abscess formation). Simultaneously with œdema glottidis or independently of it, an edematous infiltration of the submucosa is occasionally seen on the dorsal aspect of the trachea.

Symptoms. The development of inflammatory edema of the glottis takes place very rapidly; the symptoms of stenosis of the larynx appear within one quarter of an hour or within a few minutes. The animals are attacked by rapidly increasing difficulty in respiration; the inspiration especially is very forced and takes place with a marked sinking in of the intercostal spaces and of the other yielding structures of the thorax; it is accompanied by a whistling, rattling sound; expiration however is comparatively easy. The animals betray great anxiety, the eyes are staring, the eyeballs protrude, the mucosae become cyanotic, the superficial veins are enormously congested, the skin is covered with perspiration, the pulse is small and rapid. If the condition becomes worse, the animals usually succumb, generally after a preliminary fall in temperature and with convulsions.

In congestive edema the fully developed clinical picture is similar; however, its development takes several days and remissions are often noticeable.

Diagnosis. Croup of the larynx can be distinguished from primary acute edema glottidis because in the former case stenosis of the larynx occurs much more slowly and in connection with fever. Cough is characteristic. Secondary acute edema glottidis after a longer course of a primary disease leads either suddenly or gradually to stenosis of the larynx; the more chronic cases are distinguished from croup by the existence of the primary disease and by the absence of fever. Laryngeal stenosis due to lodgment of foreign bodies, animal parasites or pediculated neoplasms cannot be distinguished from edema of the glottis except by the history of the case.

To avoid mistakes, the larynx should, in smaller animals, be inspected with the unaided eye, in horses, and cattle with the electric rhinolaryngoscope; also by palpation of the larynx with the hand introduced through the pharynx provided it can be done in the presence of the existing dyspnea.

Treatment. If there is danger of suffocation, tracheotomy should at once be performed, which will immediately alleviate the difficulties of respiration, and will be followed by recovery in primary cases or in cases with moderate inflammation, provided that there is no complicating edema of the trachea. Where, in smaller animals, the symptoms are not so threatening, one may try the ingestion of small pieces of ice, the inhalation of water vapors, painting with astringents, or

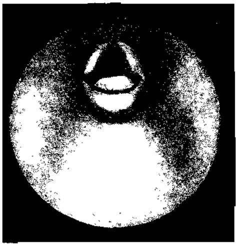
nunction with gray ointment. Nain succeeded in one case in ausing the disappearance of edema of the glottis by repeatedly pulling out the tongue of the patient.

The primary disease must, of course, be properly treated.

4. Neoplasms of the Larynx. Tumores Laryngis.

Occurrence. Tumors of the larynx are comparatively rare among domestic animals; they are encountered most commonly n the larynx of horses and cattle. Cysts develop occasionally n horses, rarely in cattle, on the anterior surface of the piglottis, exceptionally under the cricoid cartilage; they someimes attain the size of a hen's egg and are composed of a vall of several layers surrounding a mucoid mass. Fibromata, ipomata, carcinomata, melanomata, lymphosarcomata, are exeedingly rare. Van den Eeckhout observed stenosis of the arynx in a horse due to an enchondrosis of the arytenoid artilages. The round, pyriform or nodular hypertrophies of he mucosa (hyperplasia polyposa mucosae laryngis), occasionlly seen in the course of chronic laryngitis, are also to be menioned here.

Clinically similar to true neoplasms are tuberculous swellngs seen not infrequently in cattle and exceptionally in dogs Cadiot). These tuberculous masses may reach the size of hen's egg; they are sometimes pediculated, generally situated behind the lower segment of the vocal cords; and when the process extends they may spread to the outside of the larynx. Actinomycomata develop generally between the base of the epiglottis and the rima glottidis and they spread over the surface.



'ig. 110. Actinomycosis in the larynx of a cow; laryngoscopic view.

Symptoms. Neoplasms arising from the epiglottis cause difficulties in respiration and deglutition; tumors developing in other parts of the larynx give rise to stenosis sounds and to difficulties in respiration. In either case occasional or periodic attacks of cough occur. Tumors f the epiglottis interfere with respiration, particularly during rinking, while other tumors of the larynx, unless pediculated, roduce a gradually increasing dyspnea, which comes on during xercise only in the early stages. Pediculated tumors, on the ontrary, produce attacks of dyspnea, since they are aspirated om time to time into the cleft between the vocal cords. Ac-

cording to the seat and the mobility of the neoplasm, either inspiration or expiration or both may be accompanied by difficulty and by a rattling or a whistling sound. The dyspnea which comes on in attacks, suddenly disappears or diminishes considerably; i. e., when the neoplasm becomes displaced out of the air channel by a powerful attack of cough. Holterbach observed complete aphonia in a head of cattle with a tuberculous tumor in the larynx. Nasal discharge, sometimes streaked with blood, is not uncommonly present. Tumors situated in the anterior portion of the larynx in smaller animals can be immediately seen; in horses, however, the inspection of the larynx must be made with the rhinolaryngoscope (Fig. 110), or they may be felt in horses and cattle with the hand introduced into the pharynx. In horses a preliminary tracheotomy is indicated to avoid the danger of suffocation, or the preparations should be made for it. A swelling in the external aspect of the larynx is seen very rarely, and then only in laryngeal tuberculosis.

Diagnosis. The presence of tumors in the larynx can be ascertained beyond doubt only by an internal examination of the larynx; suspicion is, however, strongly aroused by the afebrile, chronic course, by dyspnea, coming on in attacks or gradually, with increasing intensity, occasionally accompanied by difficulties in deglutition, by stenosis sounds, by convulsive paroxysmal cough which sometimes relieves the dyspnea, and finally by non-tender swelling adherent to the skin on the outside of the larynx, with the signs of laryngeal stenosis. In cases of tuberculosis of the larynx, tubercle bacilli are found in the mucus from the pharynx and larynx, except in tuberculous fibromata, in which case the findings are often negative (Schlegel). In a case reported by Schmidt in a horse no tubercle bacilli were found in the nasal discharge. In suspicious cases one may, if necessary, perform laryngotomia intercricothyreoidea (Plósz).

Treatment. The difficulties of respiration can be relieved only by operative procedure. Pediculated neoplasms, or such at least as are not spread out over a large surface, may, after a preliminary tracheotomy, be removed with the écraseur introduced through the pharynx. (Almy removed cystic tumors of the epiglottis in this manner, in two horses.) The extirpation of flat, spreading neoplasms, or of those situated more posteriorly, requires laryngofissure. In actinomycotic tumors internal treatment with potassium iodide may be tried.

SECTION III.

DISEASES OF THE BRONCHI.

1. Bronchial Catarrh. Catarrhus Bronchialis.

(*Bronchitis Catarrhalis.*)

Bronchial catarrh consists in an inflammation extending to a variable extent into the tissue of the mucosa. It may either affect the larger bronchi (macrobronchitis) or only the finer bronchial branches (microbronchitis, s. bronchiolitis, s. bronchitis capillaris), or it may extend all over the bronchial tree (bronchitis diffusa). Catarrh of the larger bronchi is usually associated with catarrh of the trachea and larynx.

Occurrence. Bronchial catarrh is one of the most common affections of domestic animals and is seen in all kinds of animals, particularly in horses, dogs and cattle. Sometimes it occurs in epizootic form.

Etiology. Taking cold plays an important rôle in primary **acute bronchial catarrh.** Cold and wet weather, north and east winds, staying in the open air, pasturing during cold nights frequently give rise to colds, and therefore many animals are affected simultaneously in spring and fall. The detrimental influence of cold under these circumstances, and that of drafty air affects especially very young or very old, overheated animals or animals which have been robbed of their natural protection by shearing. Horses are therefore frequently affected after hard driving, dogs after hunting, sheep after shearing.

The inhalation of contaminated air is a further cause of bronchial catarrh. Especially dangerous in this respect are finely divided or gaseous bodies, because they can penetrate into the deeper parts of the bronchial tree. Lubenau's experiments have shown that vegetable dust can penetrate deeply and remain for a long time in the bronchial lumen. Metal dust is more apt to produce chronic catarrh. Spores of mould are inhaled during the ingestion of mouldy, musty or spoiled feed, and an acute catarrh may be produced in this manner. Dennhardt saw bronchial catarrh with severe cough develop after feeding plants which, growing near a factory, were covered with ashes.

Hot smoke (in case of fire) and irritating gases (see gas poisoning in battle) are also especially injurious.

Fluids or small solid bodies which may get into the air passages, while eating, may likewise irritate the mucosa more or less. Non-acrid, non-corroding and pure fluids are less dangerous since they are rapidly absorbed by the mucosa, as was shown by intratracheal injections. However, dried particles are often contaminated with saprophytic and other bacteria and they cannot easily be removed from the bronchi. Since substances are often aspirated in difficult deglutition, pharyngitis, paralysis of the pharynx or uncousciousness, these conditions also often lead to bronchial catarrh; aspiration may also occur when fluids are poured in in an awkward manner. The custom which prevails in many places, of pouring liquid foodstuffs into the gullets of sucking calves, in order to increase their weight, does not infrequently cause aspiration which may lead to suffocation, or acute bronchial catarrh may develop which is often followed by broncho-pneumonia or even by pulmonary gangrene (Cadéac).

The crushing of an egg in laying hens through careless palpation may lead to the aspiration of particles of the yolk into the lungs through the bronchi which open into the abdominal air sacs (Kitt). Larger foreign bodies (stones, heads of cereals, needles, etc.) may exceptionally get into the bronchi.

Bronchial catarrh undoubtedly also owes its origin to an infection, and it is then liable to assume an enzootic character. Under this type are to be mentioned the common contagious bronchial catarrhal affections seen in studs, among army horses, described under the designation of enzootic catarrh of the respiratory tract. Its true nature, however, is up to date not yet known (see page 574). The bacillus pyocyaneus frequently causes enzootic, purulent bronchial catarrh in young pigs and in cattle. Other forms of enzootic infectious bronchial catarrh, however, occur likewise in cattle and goats (Grimm, Martens, Lewek, Reisinger), especially in cattle which are being fattened (Koudelka). The bronchial catarrh observed by Schmidt in cattle in transit may likewise be due to an infection. (For further details see the chapter on catarrhal pneumonia.)

Secondary acute bronchial catarrh is frequent in diseases of the lungs, infrequent in catarrhal inflammations of larynx or trachea. It is seen frequently as a part of the clinical picture of specific infectious diseases (strangles, hemorrhagic septicemia, smallpox, foot-and-mouth disease, distemper, etc.).

Within the first two weeks, following a massive invasion of Ascaris larvæ, an acute bronchial catarrh may be produced by the migrating larvæ (see pages 362 and 364).

Chronic bronchial catarrh is caused by the same factors which bring about the acute form if the detrimental cause acts

permanently or if it leads to repeated attacks of the acute form. However, *even then the affection of the bronchi is more commonly* secondary in nature. Every continuous disturbance of the respiratory mechanism or of the pulmonary circulation brings about chronic bronchial catarrh, because not only deficient ventilation of the air passages and the decomposition of mucus due to it but also stasis of blood will cause a disturbance of nutrition of the mucosa. Consequently a bronchial catarrh is almost always seen accompanying chronic pulmonary emphysema, chronic interstitial pneumonia, organic heart disease, and chronic infectious diseases affecting the lungs (tuberculosis, glanders, actinomycosis, etc.). Animal parasites may also produce chronic bronchial catarrh (see lungworm disease).

Predisposition. Particularly predisposed to bronchial catarrh are very young, or on the contrary, very old animals, pet animals, anemic or cachectic animals.

Pathogenesis. *The mucosa of the bronchi* possesses protective features which will prevent noxious effects of external influences within certain limits. Cough excited by an irritation *of the mucosa and the epithelial ciliary movements which are* directed outward favor the expulsion of particles and microorganisms out of the trachea and bronchi which have gained *access to the bronchial tree. The covering of the mucosa* with mucus protects the latter against the immediate effect of substances which have gained access, while the numerous lymph follicles that are situated in the wall of the bronchi favor the destruction by phagocytosis of small elements which may have penetrated into the bronchial wall itself. The very smallest bronchi lack these protective structures; they are, however, sufficiently guarded by the larger bronchi situated anteriorly to them. The protective agencies become, however, insufficient if the noxious insult has been very intense, if the protective apparatus has been paralyzed, or if the detrimental stimulus travels along the blood current. Whenever the harmful agencies have gained the upper hand against the protective apparatus, the blood vessels in the mucosa become dilated, the latter swells and an increased production of mucus occurs, the substance of the mucosa becomes infiltrated with a sero-cellular exudate which also collects in the lumina of the bronchi. The substances acting as inflammatory irritants get in the further course into the pulmonary tissue with the lymph current, where they again excite an inflammatory process. If inflammation lasts for a longer time, it leads to an increase of connective tissue and to a reduction of elasticity of the bronchial wall. If the catarrh is limited to the large bronchi, the current of air in the bronchi is hardly affected, and catarrh at most gives rise to accelerated and shallow respiratory movements, by irritating the sensitive terminal filaments of the vagus nerve. In bronchiolitis the lumen of the smaller bronchi is greatly narrowed or even obstructed; the result is a diminution in the surface available for

the gas-exchange, the insufficient amount of oxygen and the excess of carbonic acid in the blood causing irritation of the respiratory center and labored breathing. A possibility exists that the process extends to the parenchyma of the lung.

Anatomical Changes. In acute catarrh of the larger bronchi (macrobronchitis catarrh. ac.), we find the mucosa reddened uniformly or in patches, either everywhere or only in some branches, sometimes studded with small dark red extravasated blood, more or less swollen, loosened and covered with mucus or with a more purulent secretion. The size of the mucous glands is from that of a millet seed to that of a hemp seed and they discharge translucent drops of mucus upon pressure.

Catarrh of the finer bronchi (bronchitis capillaris, s. bronchiolitis, s. microbronchitis catarrh. ac.), can frequently be recognized on autopsy by the fact that the lungs do not collapse well after opening the thorax, since the air cannot escape from the bronchioles which are filled with an exudate. Emphysema of the margins of the lungs (emphysema vicarians) has the same significance, and also the wedge-shaped, dark, sunken-in areas of the consistency of meat (atelectasia), while similar but firmer areas which protrude above the general pulmonary surface indicate an involvement of pulmonary tissue (bronchopneumonia). Mucous or purulent drops containing more or less numerous fine air bubbles are seen on the cut surface.

In chronic catarrh, the bronchi likewise contain a serous, mucous or purulent, occasionally a caseous, exudate. The mucosa, however, appears grayish red or brownish, unequally thickened and sometimes, on the contrary, pale and thinned out. The *connective tissue proliferation may in spots become so* intense that villous excrescences are formed in varying degrees. Sometimes, tough nodules of the size of a millet or pea, are seen outside of the bronchial wall, but in connection with it, which may contain caseous material (peribronchitis nodosa). These processes may be accompanied by a proliferation of the interstitial pulmonary connective tissue, i. e., by an interstitial pneumonia. According to Hintze this condition seems to occur not infrequently in cattle.

In the deeper portions of the lungs, especially in the anterior and lower parts, bronehiectasias are formed occasionally which are variable in shape (b. cylindriformes, fusiformes, sacciformes, etc.), and which, in larger animals, may assume the size of a fist. They always contain an abundant dirty-grayish, mucopurulent secretion, which is occasionally fetid.

In aspiration-bronchitis, in the presence of bronchiectasias, *oeeasionally also in simple chronic bronchial catarrh*, the exudate and later on the bronchial wall itself decompose with the development of a dirty-greenish color and of a fetid smell (bronchitis putrida). In aspiration-bronchitis particles of food

and other foreign bodies are usually found in the bronchi. The bronchial glands show acute or chronic swelling.

Symptoms. An invariable symptom of **acute catarrh of the larger bronchi** is cough. It is short, dry at the beginning, as long as there is no secretion in the bronchi, and later moist, with the increase and liquefaction of the secretion; then masses of secretion are expelled through the mouth or nose. Cough sometimes occurs in paroxysmal attacks. Respiratory difficulty occurs only whenever the process is more extensive, especially in the initial stage. Percussion of the thorax shows nothing abnormal; auscultation reveals various râles. At the very beginning, before any secretion whatever has occurred (bronchitis sicca), these râles are absent, and one may perhaps only hear a rough, vesicular breathing sound, but râles appear on the second or third day and remain present throughout the whole course of the disease. The râles are sharp and crackling as long as the secretion is thick and tenacious; they become more dull, moist and more numerous after the secretion has become more abundant and more fluid. Coarse and dull râles suggest, to a certain degree, the involvement of the larger bronchi, while high fine râles speak for involvement of the small bronchi. Very loud strong noises indicate, in general, an affection of the more superficial parts, those which are less audible disease of the deeper portion. Sometimes the sounds are so pronounced that they can be heard in the vicinity of the patient without auscultation and the râles arising near the surfaces may be detected by the hands placed on the surfaces (fremitus bronchialis). Commonly also such other sounds as whistling, hissing, spinning are excited by the vibrations of lamellæ of tenacious masses of secretion.

Nasal discharge, which is often present also in cattle, has generally a grayish-white mucous or mucopurulent appearance. It contains, at the beginning, few formed elements; in the further course, however, we find cell detritus, ciliated epithelia and many pus corpuscles.

The disease is generally initiated by a febrile elevation of temperature; enzootic cases which are due to an infection are particularly characterized by elevations reaching to 40° and 42° C. Sometimes a fall of temperature occurs as early as the second, generally on the third day. Increased pulse-beat, dullness and lassitude are likewise observed.

The bronchitis of sucking calves, which is due to aspiration of liquid food poured into the gullet, gives rise to convulsive attacks of cough and suffocation with decided dilatation of the nostrils, anxious look and rapid, labored respiration. If the trouble increases rapidly death may occur in a few minutes; in other cases a bronchial catarrh develops followed by broncho-pneumonia which terminates fatally. Recovery is observed occasionally.

The symptoms of **acute catarrh of the finer bronchi** are usually preceded by the signs of macrobronchitis, the clinical picture of the former rarely coming on immediately; the cough, which is likewise always present, is weak at the start and may persist until the end, accompanied by little if any expectoration. The respiration is accelerated and forced especially during expiration; sometimes paroxysmal attacks of suffocation are observed.

The *percussion sound is sometimes highly resonant toward* the boundaries of the lungs, and in such cases the pulmonary boundary is usually displaced backward. This may also be so pronounced that the boundaries as mapped out by percussion may be in a line with the costal arches (observed by Marek in horses and dogs). Atelectatic portions of the lung rarely produce dulness on percussion, and whenever such is distinctly present it usually indicates that a bronchopneumonia has occurred.

Auscultation demonstrates fine râles (high pitched), sharp crackling sounds or crepitant râles, often in connection with other sounds. The vesicular breathing sound is accentuated in many places, in others less distinct or absent, in consequence of the permanent or temporary closure of some bronchial branches with masses of secretion.

Bronchiolitis is generally accompanied by fever lasting several days or persisting throughout the whole course of the disease. The pulse is accelerated, there is dullness; lassitude and marked diminution of appetite are also always present.

In bronchial catarrh of birds, cough and hastened respiration with râles and rhonchi are observed; the respiratory sounds can be heard from a distance and are emphasized by flying. A marked dyspnea points to involvement of the lung tissue.

Chronic bronchial catarrh develops in some instances from acute catarrh. As a rule, however, it arises very insidiously and develops slowly from the start. For a long time only cough is observed. It comes on at first during exercise or after the inhalation of cold air or after the effect of other irritating factors. Generally drawn and moist, rarely painful, it appears occasionally in longer paroxysms, eventually with the expulsion of a thick mucous or mucopurulent secretion. The respiration is not interfered with in the beginning stage; later on, however, it becomes forced, and work or rapid motion bring about sneezing. Similar catarrhal sounds are heard as in acute catarrh, but dry râles and particularly purring and whistling sounds are more frequent; often these become audible only on deep breathing, after motion, or after both nostrils have been covered for a space of 30 to 45 seconds. Fever appears only during acute exacerbations or in the course of the febrile primary disease. The general condition sometimes remains undisturbed for a long time; but if the catarrh exists for several months emaciation and anemia are seen, particularly in older animals.

The bronchial catarrh which is frequently seen in cattle differs from the above-described picture only in this respect that the wheezing, whistling, and purring sounds and crepitation, are best heard at the portions of the lung covered by the shoulders. One can also demonstrate tenderness of the anterior intercostal spaces. The respiration is variably affected according to the duration and is either forced or accelerated. The general condition and the production of milk remain undisturbed. In some cases, bronchial catarrh is followed by bronchopneumonia (see there). In isolated cases the bronchial catarrh is followed by bronchopneumonia.

For details about bronchitis scleroticans of horses, as described by Grüter, *see the chapter on chronic interstitial pneumonia.*

Putrid bronchial catarrh is characterized by a peculiar, disagreeable, sweetish smell of the exhaled air, or of the occasional nasal discharge which is particularly abundant after paroxysms of coughing, and by a dirty discoloration of the nasal secretion. Otherwise the symptoms are the same as in other forms of *bronchitis; however, gangrene of the lungs frequently follows* upon putrid bronchitis. In very rare cases one is able to obtain tympanic sounds on percussion, in some part of the lower thoracic region bronchial or amphoric respiratory sounds, or possibly metallic râles as signs of an existing bronchiectasia.

Course. Acute catarrh of the large bronchi generally runs a course of two to three weeks; but occasionally recovery takes place after a few days. Bronchiolitis, however, is characterized *by a slower course and is frequently accompanied by bronchopneumonia*, which not uncommonly leads to a fatal issue; bronchiolitis, however, may alone be the cause of death.

Chronic catarrh always lasts several weeks and may last a number of months and even years. This depends upon whether the catarrh is a *primary* or a *secondary* affection, and under what conditions the patient lives. In protracted cases, acute exacerbations are occasionally seen, also attacks of suffocation, and these may lead to a great impoverishment of the condition of the patient.

Diagnosis. The diagnosis of bronchial catarrh meets with *no difficulties, the presence of râles or of whistling or purring* sounds being characteristic. One also hears râles in edema of the lungs and in pulmonary hemorrhage, but these affections come on suddenly with very grave symptoms and generally with a foamy or hemorrhagic nasal secretion. The character of the râles *and the* degree of respiratory difficulty point to the affected bronchi, while the onset and course of the disease indicate its acute or chronic character. It is, however, sometimes difficult to determine whether a bronchial catarrh is primary or secondary in character. Only a very careful examination of all organs can secure against error. The absence of symptoms pointing to any other disease speaks in favor of a primary

bronchial catarrh, and the absence of metallic râles, aside from the rare cases of bronchiectasias, as well as the generally favorable course, likewise speak in favor of primary catarrh, while *high fever and great difficulty in respiration point to an affection of the lungs.* However, catarrh of the finest bronchioles also leads to symptoms similar to those last mentioned, so that it is sometimes difficult to decide whether we are dealing with a simple bronchial catarrh or whether catarrhal pneumonia is already present.

Prognosis. Idiopathic catarrh of the larger bronchi is in general a benign affection. Catarrh of the finer bronchi, however, may in itself lead to suffocation, or it may become complicated with catarrhal pneumonia. Since experience has shown that this form of catarrh preferably affects very young or very old and debilitated animals, the prognosis in such animals is always dubious and is decidedly unfavorable in the presence of intense dyspnea and high fever. Diffuse bronchitis, which is seen after conflagrations, is likewise of very grave significance. Enzootic bronchial catarrh usually takes a favorable course and only comparatively rarely leads to death of the animals, usually after a complicating pneumonia or pleuritis has developed; even these latter complications do not exclude the possibility of recovery. In enzootic bronchial catarrh of cattle of commerce Schmidt observed a mortality of 30%.

The prognosis of secondary bronchial catarrh depends upon the nature of the primary disease.

Treatment. The principal conditions for a prompt recovery from bronchial catarrh are, that the air shall be pure, not agitated, and evenly warm. The patient should therefore be kept in uniformly warm, clean places which are ventilated freely. The required humidity of the atmosphere can be secured by evaporating water in a kettle, by hanging up moist clothes or by spraying water with an atomizer. Working animals must be kept at rest during the course of the disease. The diet requires regulating in so far as the patients should be given easily digested food which is free from dust and contains much water or is moistened with it; the drinking water should be pure.

Narcotic remedies, as they are employed in laryngeal catarrh to ease the cough, are indicated only at the onset of the trouble, if the cough is frequent and distressing, or in dry bronchitis. If the secretion is copious, as is manifested by the occurrence of râles and other catarrhal sounds, no attempts should be made to suppress the cough.

Scant or viscid secretions are removed with difficulty, especially from the smaller bronchi, by the motion of the ciliated epthelial cells and by the cough; it is therefore advisable to liquefy them by means of expectorants, either alone or combined with soothing remedies (root and juice of liquorice; root and

syrup of althæa). This indication is met well by certain salts which are eliminated in part on the bronchial mucosa and stimulate an increased elimination of water, and by alkaline carbonates which liquefy phlegm. Such remedies are chloride of ammonia (8-15 gm. for large animals, 0.2-1.0 gm. for small; birds are to be given table or coffeespoon doses, 3 or 4 times daily, of Ammon. chlorid. 0.5, Melis. 0.5, Aquae foenic. 50.0); sodium chloride is given to larger animals, mostly in the form of artificial Carlsbad salt (cattle 20-200 gm., horses 10-20 gm.), to dogs in the form of a saline mineral water; bicarbonate of soda is administered in like doses; of iodide of potassium the doses for horses and cattle are 5-10 gm., for dogs 0.25-1.0 gm. In a horse with bronchiolitis, Hermann had good results from repeated subcutaneous injections of iodipin (40-50 gm.). For their stimulating action upon the secretions, emetic remedies in doses which do not cause vomiting, are of value; root of ipecacuanha (to smaller animals tablespoon doses, 3 times daily, of an infusion 0.5-150.0); hydrochlorate of apomorphine (0.005-0.01 :50.0, if necessary combined with 0.05 gm. of hydrochlorate of morphine; one spoonful every 3 hours for dogs); hydrochlorate of pilocarpine (for horses and cattle, 0.1-0.3 gm.; for dogs 0.005-0.01 gm., subcutaneously); tartar emetic (cattle 1-5 gm., horses 0.5-2.0 gm., sheep, goats, hogs 0.1-0.5 gm., dogs 0.01-0.05 gm.); Stibium sulfuratum aurantiacum (cattle 10-20.0 gm., horses 5-15 gm., hogs, goats, sheep, 1-5 gm.); senega root (in a decoction 10:150.0 for dogs).

If excessive secretion, especially in the bronchioles, causes dyspnea, emetics are indicated in smaller animals, so as to promote the removal of the secretions; this will be accomplished because the vomiting increases the pressure in the respiratory passages and, following this, produces an increased air current. Remedies of this class are muriate of apomorphine (for dogs 0.005-0.01 gm.; for cats 0.002-0.005 gm., subcutaneously); veratrine (for hogs, 0.03 gm. dissolved in alcohol, subcutaneously); tartar emetic (dogs 1:100.0 gm., tablespoonful every 15 minutes; sulphate of copper (dogs 1:40.0, tablespoonful every 15 minutes); ipecac (1-2.0:15.0 of water), etc.

If the excessive production of thin liquid secretions causes dyspnea, it may be relieved temporarily by subcutaneous injections of atropine sulphate (to horses and cattle 0.03-0.05 gm., dogs 0.002-0.01 gm.).

The inhalation of salt solution, and of astringent and disinfecting solutions (page 528) is of no practical value because only a comparatively small portion of the inhaled vapors or particles of the fluids enters the deep air passages, most of them being arrested in the nose and pharynx. The favorable action of these inhalations consists primarily in rendering the inspiratory air very humid. Oxygen inhalations relieve or diminish the dyspnea which is severe in extensive bronchiolitis.

Of subordinate practical importance are also intratracheal

injections of remedies which are intended to loosen the phlegm or which have an astringent and disinfecting action. The systematic application of these injections meets with dfficulties, and the fluids which are injected into the trachea reach only the larger bronchi of the anterior lung portions; watery solutions are absorbed rapidly. To insure better success, it would be necessary to inject large amounts of fluids, or emulsions of oils; appropriate drugs are sod. bicarb. (2-3%), zinc. sulph. (½%), Argent. nitrat. (0.1%), Lugol's solution (1:5:100-200); oil of turpentine (with equal parts of olive oil). Aqueous solutions are injected in larger animals in amounts of 20-50 gm., smaller animals 2-5 gm., oil emulsions 15-20 gm. at one operation. Tincture of opium may be added to all of them (1-2%) in order to reduce the great irritability.

In putrid bronchial catarrh, little can be done with inhalations of finely sprayed solutions of corrosive sublimate (1:2000), of 2-3% solution of creolin or 1-2% solution of carbolic acid, but intratracheal injections of disinfectant solutions exert a manifest, if often transitory effect. In personal observations, injections of 100 cc. of a 1% solution of carbolic acid twice daily were sometimes of value in horses. Masson & Vazeuz were successful with creosote (1 gm. creosote, 40 gm. alcohol, 40 gm. aq.; of this mixture 20 cc. twice daily). Intratracheal injections of a 4% solution of formol sometimes produces a protracted and distressing cough.

Warm, moist applications to the thorax may be of use in all cases of extensive bronchial catarrh if there is no severe dyspnea; if this is present, the applications are apt to increase it.

The general condition must be watched in very young and in very old animals and stimulants must be given in the presence of debility (camphor, ether, alcohol, caffeine).

Bronchial Croup (*Bronchitis crouposa*). Cattle and sheep suffer from a croupous inflammation of the bronchial mucosa under the same conditions which bring about croup of the larynx, or it may be secondary to nasal or laryngeal croup. In idiopathic bronchitis crouposa the mucosa of all bronchi or of the majority of them is covered by *tubular masses of fibrin*. *These masses interfere more* or less with the exchange of gases and they close the finer bronchi completely.

Hence the symptoms of this affection consist in a very grave dyspnea which reaches a high degree, either suddenly or more rarely after a preliminary fever which is not well marked. One hears rattling, whistling and purring sounds over the thorax. They are as a rule very loud and may be felt as a pectoral fremitus. The disease is recognized in its true nature only after the patients have coughed up croupous membranes, the source of which is made obvious by their tubular shape and branching arrangement. This does not, however, occur in all cases, because the animal may suffocate without any expulsion of membranes or the latter may not be thrown out but may be swallowed and so escape notice.

The disease may reach its full development within 2 to 3 days

and may then lead to suffocation, especially in young animals, while older and stronger animals may occasionally recover after the expulsion of the membranes.

In primary bronchial croup in young cattle, the animal suddenly shows difficult breathing, great fright and restlessness without having previously shown other disturbances. The symptoms suddenly disappear after the expulsion of branching, fibrinous casts which are spirally twisted at the end. During the expulsion the animals exhibit movements of coughing and symptoms of asphyxiation.

The treatment is similar to that recommended for laryngeal croup; tracheotomy, however, is not necessary in primary bronchial croup.

[The disease of cattle and sheep described above is similar to one occurring in man and now generally called bronchitis fibrinosa. It is, however, a very chronic affection in man, which usually leads to a fatal issue. Translator's note.]

2. Narrowing of the Large Bronchi. Bronchostenosis.

Etiology. Stenosis of the larger bronchi may be caused especially by the accidental introduction of foreign bodies, ears of grain, fir-cones, pieces of bone, cartilage, wood, needles, etc., as they have been found repeatedly in the air passages of horses, cattle, swine and carnivora. (Szabo found a mouse in the air passages of a hog that had suffocated suddenly, which got there accidentally while the animal had been rooting.) Other causes are narrowing of the bronchial lumen by croupous pseudomembranes or compression of the bronchi by enlarged or suppurating bronchial or mediastinal lymph glands, aneurysms of the aorta, tumors of the lungs or of the mediastinum. Ball found concrements (bronchioliths) in the bronchi of a cat suffering from paroxysmal cough and dyspnea; these consisted of phosphates and carbonates. In an old horse he found a pneumolith in a pulmonary cavity.

Symptoms. Narrowing or complete obturation of a larger bronchus diminishes the exchange of gases in the corresponding portion of the lung or prevents it entirely and causes difficulties in respiration, which come on suddenly in the case of the introduction of a foreign body, but more gradually in case of compression by a tumor. Particularly the former condition leads to convulsive paroxysms of cough. Difficulty of respiration is generally of a much graver form in consequence of sudden closure than it is in a stenosis which has developed gradually. The stenosis is usually unilateral, it lessens the excursions of the corresponding half of the thorax and causes a sinking in of the yielding portions of the thoracic wall in inspiration. A stenosis sound similar to that heard in laryngeal stenosis may likewise be observable.

The percussion sound remains unchanged as long as there is not a complete obstruction. While in complete obstruction, according to the experimental results obtained by Heller, com-

plete atelectasis develops in from 2 to 2½ hours in that part of the lung contiguous to the obstructed bronchus. A dullness of the percussion sound is produced over such areas and all respiratory sounds are absent. The vesicular breathing sounds are absent over the affected half of the thorax, or at least weakened, and are intensified over the other portions of the chest. Over the affected parts are heard occasionally rattling or whistling sounds.

In stenosis caused by a foreign body the exhaled air later on becomes fetid and the temperature rises. In stenosis caused by compression one occasionally also observes edematous infiltration on account of the simultaneous compression of larger venous trunks, paralysis of the larynx due to compression of the inferior laryngeal nerve, disturbances of deglutition due to compression of the esophagus, and in ruminants chronic bloating.

Treatment. Since successful treatment is impossible, the animals ought to be utilized by early slaughtering.

3. Asthma bronchiale.

(Asthma spasmodicum, Asthma nervosum.)

Bronchial asthma, or simply asthma, is a term applied in human medicine to attacks of severe dyspnea, which occur at irregular intervals and are produced by temporary narrowing of the bronchial lumen, through convulsive contractions of the bronchial muscles; this is followed by acute emphysema and by a peculiar bronchial catarrh characterized by rattling and whistling sounds, by the occurrence of the so-called Curschmann's spirals (spiral threads of mucus), by immense numbers of eosinophile cells and of needle-shaped Charcot-Leyden crystals contained in the viscous mucous expectoration. In the intervals between attacks, all symptoms of disease are usually absent. The disease is generally looked upon as a vagus-neurosis occurring in consequence of a direct irritation of the vagus by compression (swellings, enlarged lymph nodes), or by reflex irritation in the course of bronchial catarrh, certain affections of the nasal and pharyngeal mucosa and the gastric mucosa. According to Manoiloff, bronchial asthma may be viewed as a temporary anaphylactic phenomenon produced by Charcot-Leyden crystals, the products of disintegration of a foreign protein in the respiratory passages, the source of which is unknown. A nervous disposition also plays a part. The asthmatic attacks may be successfully combated by subcutaneous injections of adrenalin and atropin, by subcutaneous injections of hypertonic sodium chloride solution (18%), or by the internal administration of urethane.

Asthma in domestic animals has been described repeatedly, but the correspondence of these cases with true asthma is not established. The expectorations were not examined for Curschmann's spirals, eosinophile cells and Charcot-Leyden crystals. In the interpretation of the cases,

transitory stenosis of the air passages, due to anatomical changes, were not considered, and finally it was not taken into consideration that attacks of severe dyspnea are not infrequent in permanent stenosis of the respiratory passages, in pulmonary emphysema, in the course of chronic uremia (Asthma uræmicum), and of chronic heart disease (A. cardiacum). Nevertheless the occurrence of true bronchial asthma in domestic animals cannot be denied *a priori*.

In the six cases of supposed bronchial asthma in horses, reported by Penberthy, the sudden attacks of dyspnea disappeared in 2 to 24 hours suddenly after the discharge of bronchial secretion by severe attacks of coughing. A lasting improvement did not materialize, and in two of the horses the clinical signs of pulmonary emphysema were demonstrated. Speiser observed paroxysmal dyspnea in five horses. Grebe interpreted a case as asthma dyspepticum, in which a horse had been affected for ten days with dyspnea, occurring each time food was taken; the inspirations were labored and accompanied by a soft, whistling sound; the dyspnea persisted for one-half to one hour after eating. During the examination it increased to such a degree that the horse was in danger of falling. In the intervals, no respiratory difficulties existed; an injection of eserine was followed by complete recovery.

In cattle, bronchial asthma was described first by Boer, who noted in two heifers a whistling inspiratory sound during attacks. Royer saw asthmatic attacks with extreme dyspnea in epizootic distribution, in very foggy regions; it is probable that he was dealing with interstitial pulmonary emphysema.

4. Lungworm Disease. Bronchitis et Bronchopneumonia verminosa.

(*Verminous bronchitis; Verminöse Lungenphthise, Strongyluskrankheit, Lungenwurmseuche, Lungenwurmhusten* [German]; *Strongylosis pulmonum; bronchite et bronchopneumonie vermineuse, Strongylose bronchopulmonaire* [French].)

Lungworm disease is an affection which is usually enzootic, even epizootic. It occurs in the form of a bronchitis or bronchopneumonia of variable extent and is caused by lung palisade worms (strongylides).

Historical. Epizootics among animals caused by worms were first described in the middle of the eighteenth century. They attracted more universal attention, however, only after another hundred years. The following have especially studied this affection: Davaine (1860), A. Koch (1883), Neumann (1888), A. Müller (1889), Tapken (1891), M. Schlegel (1899), Jeanmaire (1900), Docter (1907), Joest (1908), Schoettler (1911), and others.

Occurrence. The disease occurs in all countries, usually among animals which are kept in marshy pastures or in pastures which are frequently exposed to inundations or situated

in lowlands. Accordingly, it is predominantly a pasture disease, and sporadic cases may occur on previously noninfested pastures if, through a high ground-water level or other conditions, such pastures have on them much standing or flowing water. The disease sometimes occurs during stable feeding, especially in hogs, more rarely in cattle. Among domestic animals, sheep and goats are most commonly affected, in some neighborhoods also swine; more rarely affected are cattle and camels; horses, asses, cats, dogs and rabbits only exceptionally. Lungworms are very frequently found among some wild mammals such as deer, roes, chamois, boars and hares. As a herd disease, it is generally only met with in sheep and goats, especially among younger animals, and from time to time it causes serious losses, particularly after wet summers. The enzootic appearance among swine herds is rarer; it is still less common among grown cattle and calves. Enzootics of the disease have occasionally been observed among camels. The disease appears not infrequently as a very destructive epizootic among the wild animals mentioned above.

According to Carnet half of all sheep and one-third of the cattle in Morocco succumb in some years. The disease occurs epizootically in South Oranais, according to Ben Danou, and is called "Reuch" by the natives, and when disease of the lung is simultaneously present they call the affection "Reuch-el-riya." Three-fourths of all sheep (many millions of animals) succumbed in Buenos Ayres from 1883 to 1886. The disease is quite destructive in some parts of Germany (on the lowlands of the Weser and the Jade rivers) among cattle and calves (Tapken). The disease is also widespread among swine in some provinces in Russia, and of the swine slaughtered 14% were found affected in St. Petersburg, 21% in Moscow and 48% in Riga (Schultz). Heavy losses among sheep were caused in Hungary in the year 1889.

In slaughtered animals lungworms are found frequently which have, however, not affected the health of such hosts unfavorably. Hertwig gives the following figures for the Berlin slaughtering plant for 1887-88. Among 275,049 sheep, 788 or 3%; among 419,848 swine, 3,237 or 7%. In the Budapest stock yards there were found from 1889 to 1903, among 90,883 sheep, 3,082 or 3.4%; and from 1902 to 1903, among 474,401 hogs, 320 or 0.067%.

Etiology. The lungworms (lung-strongylides) belong to the filiform worms (Nematodes) and are long, thin, smooth worms; the small mouth, at the anterior end of the body, is often surrounded by three to six papillæ. The body of the male, which is always shorter, ends in a bursa which is variable in shape and which serves for grasping the female, being provided with two spicula. The posterior end of the female is straight. The genital pore is sometimes behind, sometimes in the middle of the body.

The following palisade worms have been found in the respiratory organs of domestic animals.

1. **Strongylus (Dictyocaulus) filaria** Rudolphi. A white filiform worm with rounded, bare mouth without papillæ. The male is 3-8 cm. long, the bursa has an anterior cleft; the anterior rays are double, the middle rays have two, the dorsal ones three divisions. The spicula are brown, short; moderately bent, supplied with membranous wings; female 5-10 cm. long, posterior end pointed; the genital pore somewhat behind the mid-line of the body. Ova (Fig. 111) are oval, have a delicate hyaline

membrane 112-115 μ long, the embryo is well developed within the mother animal and is liberated immediately after the eggs are deposited in the bronchi.

The worm is parasitic in the respiratory organs of sheep and goats, also in roes, in gazelles, in camels, deer and fallow-deer.

2. **Strongylus micrurus** Mehlis (Dictyocaulus viviparus, Bloch). Long slender worm with pointed ends, smooth head and mouth. Male 3.5-4 cm. long with small simple bursa which is supported by five ribs and which is supplied with powerful spicula; female 6-8 cm. long, posterior end strongly pointed, genital pore directly behind the mid-portion of the body. Ova (Fig. 112) are oval, about 85 μ long, with delicate shell, containing an embryo in the mother animal. The head end of the embryo is thicker, the posterior portion of the body is S shaped.

The parasite occurs in cattle, fallow-deer, stags, exceptionally also in horses and asses.

3. **Strongylus (Dictyocaulus) Arnfieldi** Cobbold. A white filiform worm with bare mouth. The male is 2.8-3.6 cm. long; the short bursa is lobulated laterally and caudally, the anterior ribs are forked with unequal divisions; the middle ribs branched evenly; the posterior ribs originate in a common trunk and their ends are lobulated. Spicula brownish yellow, slightly bent, with net-like marking. The female is 4.3-5.5 cm. long, the caudal end blunt, slightly bent, the vulva somewhat behind the middle of the body. The eggs are elliptical, 80-100 μ long. The embryo is fully developed when the eggs are deposited, and is liberated in the respiratory passages of the host.

Parasitic for horse and ass.

4. **Strongylus paradoxus** Mehlis (Metastrongylus apri Gmelin, Str. longevaginatus Diesing). Whitish or brown worm with conical head and six papillæ at mouth. Male 1.2-2.5 cm. long, bursa composed of two wings, the two spicula are very long, thin, each terminating in a barb. Female 2.5-5 cm. long; a spheroidal swelling in front of the pointed end; the vulva in front of the anus. The eggs (Fig. 113) are short, oval, 57-100 μ long, with thick, transparent shell; the embryo is

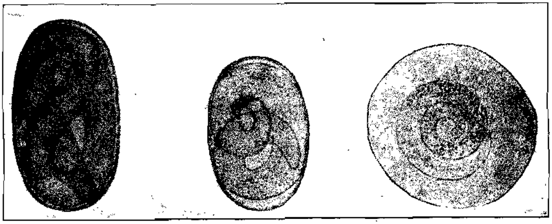


Fig. 111. Ovum of Strongylus filaria.

Fig. 112. Ovum of Strongylus micruris.

Fig. 113. Ovum of Strongylus paradoxus.

developed at the time the eggs are deposited and is liberated in the bronchi of the host; its tail end has a bulbar thickening.

Parasitic for domestic and wild hogs, exceptionally for sheep, roes and deer.

5. **Metastrongylus brevivaginatus** Railliet & Henry. Differs from the preceding worms by different shape of bursa and the short spicula, each of which ends in two barbs. Parasitic for domestic hogs.

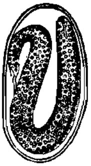


Fig. 114. Egg of Strongylus capillaris. (After Schlegel.)

6. **Strongylus (Synthetocaulus) rufescens.** Leuckart. (Str. minutissimus, Str. ovis pulmonalis). Hair-fine, thin brownish-red worm with three-lipped mouth. Male 1.8-3 cm. long, the bursa indented in front, laterally slightly curved inward; two long spicula with transverse stripes and rounded ends. Female 2.5-3.5 cm. long, blunted tail-end, the vulva immediately in front of the anus, at the base of a small elevation. Eggs oval, 75-100 μ long. At the time when they are deposited in the lung alveoli of the host, they have reached the stage of grooving, the embryo then develops rapidly and is liberated in the lung alveoli of the host. Its posterior end is provided with a hook-formed process.

Parasitic for sheep, goats, rabbits and chamois.

7. **Strongylus (Synthetocaulus) commutatus** Diesing. Brown filiform worm with bare mouth. Male 1.8-3 cm. long, the bursa very small, rounded, the posterior ribs combined; spicula broad and thick, like a sound, marbled at the upper end. The female 2.8-5 cm. long, blunt tail-end, vulva immediately in front of the anus. The eggs are grooved when deposited in the lung alveoli of the host; the embryo develops and becomes free in the alveoli; the tail is provided with a straight, lance-like

Fig. 115. Embryos of long worms (after Jerke and V. Linden and Zenneck). a, Strongylus filaria; b, Str. micrurus; c, Str. paradoxus; d, Str. commutatus; e, Str. capillaris.

process. According to Müller, this worm is identical with the Str. rufescens.

Parasitic for hares, rabbits, sheep, goats, roes, chamois.

8. **Strongylus (Synthetocaulus) capillaris.** Müller (Pseudalius *ovis pulmonalis. Nematoideum ovis pulmonale). This worm is thin* like a cobweb, cylindrical, brownish, transparent (hence called "lung-hairworm); six mouth-papillæ and pointed tail end. Male 1.4 cm. long, the posterior end corkscrew-shaped, turned in; the small bursa is supported by seven ribs which are usually folded; club-shaped upper end of spicula bears transverse chitin bristles; the posterior end is forked, and the ventral branch is shaped like saw-teeth. Female 1.9-2.3 cm. long; the vulva just in front of the anus. Eggs brownish, not grooved when deposited. The embryo develops after the eggs are deposited: its tail has a snake-shaped process and a dorsal barb; it is liberated in the alveoli and in the bronchi of the host. The mature worms invade *the lung tissue, after depositing the eggs, where they are encapsulated* and then die.

Parasitic for sheep, goats, perhaps for roes and chamois.

9. **Synthetocaulus unciphaurus,** *Railliet & Henry; occurs in sheep* and goats.

10. **Strongylus pusillus,** Müller (Synthetocaulus abstrusus Railliet). Filiform worm with bare mouth. Male 0.5 cm. long: bursa short, spicula thin, slightly curved. Female about 1 cm. long: blunt tail-end; vulva closely in front of anus. Eggs short oval, 60-85 μ long, grooving occurs after they are deposited. The tail of the embryo is provided with a hook-formed process.

Parasitic for domestic cats.

11. **Strongylus (Haemostrongylus) vasorum,** Railliet. A filiform, whitish or reddish worm with longitudinal stripes, mouth bare. Male 1.4-1.8 cm. long. Female 1.8-2.1 cm. long. Eggs 70-80 μ long, oval, not grooved when deposited by the mature animals which live in the right heart and in the branches of the pulmonary artery. They are arrested in the lung capillaries where the embryos are liberated.

Parasitic for dogs.

12. **Filaria tracheo-bronchialis canis** (Fil. Osleri, Strongylus bronchialis canis). Filiform worm, 0.5-1.5 cm. long; three-lipped mouth with three unequal papillæ; two unequal spicula in the male, vulva close in front of anus in female. Its position in the system is not *established definitely. It occurs in the wart-like tubercle of the trachea* and in its primary divisions in dogs.

13. **Trichosoma areophilum,** Creplin. Whitish yellow worm with *transverse stripes, 2.4-3.2 cm. long. In the male the bursa has two* lobes, the sheath of the spiculum is covered with small warts. The eggs (Fig. 4, page 31) *are* 68 μ long, lemon-shaped, with whitish shells. This worm has been found in the trachea and bronchi of a fox (Creplin), in cats (Müller, Neumann), in a badger (Müller), also in dogs (see page 30).

14. **Ollulanus tricuspis,** Leuckardt. A worm barely 1 mm. in length; bursa two-lobed, supported by six ribs; vulva near the anus. The mature worms live in the stomach of cats and deposit embryos, some of which invade the lungs.

The various kinds of domestic animals can be invaded by several strongylus species. Of these occur:

In sheep: Str. filaria and Str. capillaris, less often Str. paradoxus, rufescens, commutatus.

In goats: Str. filaria, commutatus, rufescens, capillaris.

In hogs: Str. paradoxus, Metastrongylus brevivaginatus.

In cattle: Str. micrurus.

In horses and asses: Str. micrurus and Str. Arnfieldi.

In dogs: Filaria tracheobronchialis and Str. vasorum.

In cats: Str. pusillus.

In rabbits: Str. commutatus, rufescens.

As to the frequency in occurrence of individual species of lungworms in domestic animals which harbor several species of strongylus, the following may be said: In sheep one finds, most commonly, according to the general view, Strongylus capillaris and exceptionally only Str. paradoxus or Str. minutissimus; not uncommonly, however, there may be more than one, and in fact all of these three species of strongylus in one and the same host animal. It is said that the disease generally takes an enzootic character if the Str. filaria is present, more rarely if Str. capillaris, and still more rarely if other species of strongylus are present. Some observations, however, point to the fact that these conditions may be changed materially, particularly in some parts. Ranke found Str. capillaris in all sheep marketed in London. Ströse found this same worm in 66%, and Schlegel in 22% of the sick sheep, while the last-named author found Str. commutatus in 33% and Str. filaria in only 4%. It is also believed that goats are infected most commonly with Str. filaria; but Schlegel found the Str. capillaris in 92% of sick goats.

Evolution of the Lungworms. The embryos which hatch soon after the eggs are deposited, or in oviparus worms those which hatch in the lumen of the bronchi after the lapse of a certain time, and also those which still remain in the eggshell, are expelled directly with the bronchial secretions into the outside world, or they get into the pharynx, are swallowed, and are voided with the feces. According to von Linden and Zenneck, only the slender embryos contained in the large eggs are seemingly capable of further development in the outside world, while the plump embryos in the smaller eggs are probably responsible for an endogenous multiplication.

A direct evolution takes place, whereby, according to the experiments of von Linden and Zennek, a number of nonparasitic free-living sexual generations may develop in sterilized soil seeded with grass seed (denied by Richter). The hatched filiform larvæ pass through their first molt in from 1 to 2 days (Strongylus filaria, S. micrurus)—that is, while still in the intestinal tract of the host (Jerke), or, at latest, after they have been voided in the feces. (According to Richter, the passage of the eggs and embryos of St. filaria through the intestinal tract has a determining significance for their future development, since the expectorated embryos soon perish or only reach the simple larval stage.) In doing so, they do not cast off the cuticula but remain encysted in the loosened skin. Under favorable conditions they pass through a second molt in from 12 to 48 hours, although the first skin is not cast until another 12 to 48 hours have passed. The larvæ remain encapsulated in the second skin and become very resistant. Varying with the outside conditions, the larvæ become infective in from 6 to 15 days after the eggs have been deposited. When such larvæ are ingested hy a suitable host, they cast their second cuticular or cystic envelopment and after two further molts reach maturity. Thus, five stages of development, separated by four molts, can be distinguished.

In passing to the free-living sexual generations, the rhabditic form acquired during molting is retained. The development of the first free-living mature generation requires from 4 to 6 weeks and the female lays only a single egg. In the following generation an increasingly larger number of eggs are laid at a time. These generations are of microscopic size (0.6 to 0.9 mm. long) and multiply rapidly at times, while at other times the multiplication is at a standstill for months. In Strongylus micrurus von Linden observed the most rapid multiplication in June, July and August; in S. filaria, in April and June. Weather conditions undoubtedly play an important role in this respect. A medium degree of moisture and favorable temperature (10° to 40° C.) are especially favorable for development, while dryness, low outside temperature, and putrefaction retard development. Accordingly, development can proceed uninterruptedly during the warmer season of the year in moist soil, in the filthy or muddy water of pools and swamps, on wet grass, and in slightly rotting manure which has retained sufficient moisture.

According to Gerlach the period of development to sexual maturity lasts at least eight weeks; however, the observation of Schlegel, that lungworm disease in goats may appear twelve to fifty-nine days after the introduction of the brood of Str. capillaris, permits the conclusion that sexual maturity may be attained in a shorter time than eight weeks. (Str. micrurus, according to Tapken, needs six to seven weeks for its complete development.) The location where the worms attain full sexual development varies more or less for different species. Str. filaria is found preferably in bronchi of medium and small size; Str. commutatus is found in the smaller bronchi and penetrates into the pulmonary tissue after having deposited its eggs; the same is true of Str. capillaris, which is found in the small bronchi and alveoli. Str. micrurus is found preferably in the medium-sized bronchi (Jöst). The usual habitat of Str. paradoxus is in the medium and smaller bronchi; but occasionally it is found in the large bronchi and even in the trachea. Other lungworms inhabit the medium and small bronchi. Trichosoma aerophilum is a parasite of the trachea and of the large bronchi, while the sexually mature individuals of Str. vasorum are found in the right side of the heart and the pulmonary artery; those of Ollulanus tricuspis in the gastric mucosa.

The power of resistance of the worms appears to be very poor directly after the embryos are set free out of the egg shell; especially embryos of Str. filaria and of Str. micrurus perish within a short time under unfavorable conditions (putrefactive processes, desiccation), while otherwise they remain alive for weeks and go through one moulting in pure water. After the first moulting, the brood, probably now in a rhabditis-like intermediary form, attains great resisting power, so that it may live in water for months (Railliet, Baillet, Cohn), and may revive after a long period of desiccation. Larvæ of Str. filaria were found alive after sixty-three hours of desiccation (Railliet), after a year (Ercolani, Piana), and those of Str. commutatus after fourteen months. Sexually mature worms and their larvæ are not very resistant if brought in contact with antiparasitic substances. Piana's investigations have shown that the larvæ are killed at once by an iodine-iodide of potash solution (1:10:100) and by a 4% corrosive sublimate solution; by vapors of iodine in one minute; by 15% solution of common salt in ten minutes; by a 10% solution of potassium iodide in fifteen minutes. Oil of turpentine, creosote and calomel apparently do not influence the larvæ, which, however, do not revive after desiccation followed by soaking in water. Absolute alcohol immobilized them for awhile, but they became motile again after some time.

Infestation usually occurs on pasture by ingestion of the larvæ attached to grass or living in water. The infestation of

the pasture, as a rule, takes place by manifestly sick animals or seemingly healthy carriers constantly eliminating larvæ with the bronchial secretions or the feces. These larvæ will survive for years and in the meantime undergo sexual multiplication. Accordingly, pastures that have not been used for years may still retain their infectivity. Pastures that have previously been free from infestation may become infested by overflows or by wild animals. According to von Linden, pastures may also become infested through chickens and by wind-borne molds. It does not seem strange, therefore, that lungworm disease becomes established in many regions.

According to the biology of lungworms, swampy, marshy and irrigated pastures and pastures subject to overflows are most dangerous, and this danger is greatly increased by continued rainy weather. Hence, on favorably located pastures and during rainy years the disease occurs epizootically over wide regions. On the other hand, outbreaks of the disease have been observed on dry and more elevated land. This can be explained by the fact that the lungworms can develop in the open in the absence of particularly favorable outside conditions and withstand drying out for years, while the usually periodic rains and dew favor their periodic development. Accordingly, the disease also occurs in dry years, and it is not absolutely necessary that the larvæ be inhaled with dust. It is not likely that the larvæ are cast upon the vegetation by heavy rains and wind. Wooded pastures that are used only periodically may also be dangerous, since they may easily become infested by large game. Under no circumstances should pastures be used soon after a rain or when they are wet with dew (in the early morning hours), since the larvæ follow the moisture and collect in the dewdrops at the tips of the blades of grass.

Infection in the barn is also possible. A porous and continually moist soil of improperly arranged hog pens, also the soil of the stables of other animals, the moist soil in the neighborhood of drinking places, stagnant water in the latter, offer a chance for the further development of embryos which are coughed up by infected animals. Indeed, lungworm disease has repeatedly been observed in young pigs which have never left the pen (Schultz, Moussu, Marek), or in cattle kept permanently in the barn (Kasparek, Scheibel). Sucking animals may occasionally infect themselves from the udders of their mothers which have become infected through contaminated straw. Exceptionally an infection may occur through dry feed contaminated with desiccated but living larvæ.

The possibility of intrauterine infection must be admitted as demonstrated by the observation of Neveu-Lemaire who found mature specimens of Strongylus filaria in the trachea of a fully developed fetus of a mother-sheep, dead of lungworm disease, and in a four-day old lamb. The larvæ taken up by

the mother animal get into the circulation, and a few pass through the placenta into the fetal circulation of lungs and bronchi.

The brood of the worms is usually taken up into the body of the host in spring at the beginning of pasturing; newer observations, however, permit the conclusion that infection may also occur in summer and even late in the fall (Schultz). According to Docter, field hares infect themselves in fall. Repeated invasions are not at all rare.

Direct contagion does not occur; experiments to infect healthy animals through the introduction of bronchial mucus containing embryos into the respiratory passages or into the stomach, or by the intravenous introduction of embryos and ova (Leuckart, Schlegel, Jean Maire), were not successful.

In spite of this, sick animals and carriers are dangerous for animals that graze with them on a common pasture or are kept in a barn with them, since the larvæ eliminated by them will soon become infective.

Susceptibility. Young animals which have at least reached an age of several weeks are preferably affected. The observation of Kasparek, according to which calves one and one-half to eight days old were affected, has remained unique. Older animals are affected more rarely in direct proportion to advancing age. Sometimes, however, sheep and goats are affected equally and independently of age, especially in infection with Str. capillaris. The clinical symptoms are, however, usually much milder in older animals.

Pathogenesis. With the ingestion of food and drink the larvæ get into the stomach, and from there, during rumination or by active wandering toward the pharynx, they get into the respiratory passages. Although now given up almost generally, the view has much to recommend it, that the eggs of worms get from the intestines into the blood, with this into the pulmonary alveoli and into the bronchi. Joest believed this mode of invasion to be possible; Neveu-Lemaire takes it to be proved by his observation, already mentioned, of intrauterine infection in two lambs. Zürn, Spinola and Csokor claim that the worm brood may also get directly into the air passages by the inhalation of dust from contaminated pools and marshes. The idea that the larvæ may get directly into the lungs with the blood current has been generally abandoned. Joest, however, believes that this mode of invasion, with a subsequent migration of the brood from the lung into the bronchi, cannot be entirely denied. Strongylus larvæ which get into the alveoli and bronchi, probably invade then the smaller bronchi, some also the pulmonary alveoli, and produce, partly by their motion, partly by their metabolic products, an inflammation of the invaded bronchi (bronchitis verminosa) or of the alveoli. Since the invasion is usually not very intense, and since the degree of inflammation

and the number and size of the inflammatory foci stand in direct relation to the amount of the worm brood, either disseminated and generally small inflammatory foci are developed in the lungs (bronchopneumonia verminosa lobularis) or the inflammation remains confined solely to the infected bronchus. During their further development, most lungworms (excepting strongylus capillaris and strongylus commutatus) try to enter the larger bronchi, where they reach sexual maturity and also cause an inflammation which, however, is mostly due to the deposited ova or the escaped embryos. Embryos may be aspirated into the smaller branches of the larger bronchus or wander there actively and after perforating the wall of the bronchioles, get into the lung parenchyma, where they produce inflammatory changes, and in this manner inflammatory foci develop in large numbers and in considerable extent (bronchopneumonia lobularis disseminata). However, the spread of worm pneumonia probably depends partly upon the auxiliary action of bacteria (Joest). According to Olt, the worm-nodes which are harmless in themselves, in roes, are complicated by croupous pneumonia which may be fatal, in consequence of secondary infection with the Bac. bipolaris septicus, or by a purulent bronchopneumonia by localization of the Bac. pyogenes in the lung. Schlegel found in goats a coexisting infectious pleuropneumonia. Lignieres called attention to the occurrence of these secondary infections. In consequence of perforation of the bronchial wall by the embryos, air may get from the bronchial lumen into the lung parenchyma. But it is questionable whether interstitial emphysema of the lungs, which is seen frequently in cattle in connection with lungworm disease, is mainly due to perforation of the bronchial wall, as claimed by Joest, since the bronchial lumen is usually obstructed by masses of exudate before perforation occurs. Paroxysms of cough due to the existing bronchial catarrh appear much more important. Sexually mature worms (Str. capillaris and Str. commutatus) invading the lung tissue or embryos (Str. vasorum and Ollulanus tricuspis) by way of the blood current give rise to the formation of miliary or somewhat larger inflammatory foci.

From the bronchi the inflammatory process usually spreads to the peribronchial tissue and even further, and the diminution of elasticity of the bronchial walls induced in this manner, gives rise to a dilatation of the larger bronchi. Occasionally some bronchi are obstructed by masses of worms and secretions, causing atelectasis of the affected portion of the lung, which again forms the basis for a bronchopneumonic focus. If embryos are swallowed in great numbers, they may interfere with digestion.

Extensive bronchial catarrh or bronchopneumonia cause distnrbances of nutrition in consequence of an increased decomposition. Linstow and v. Linden suggest that the worms secrete hemolytic poisons; but this has not been proved.

Anatomical Changes. These are quite variable according to the number of lungworms, to single or repeated invasions, to the species of the parasites, the duration of the process, etc. As a rule, airless foci are found in the otherwise normal or emphysematous and pale lung tissue, preferably in the anterior and inferior portions of the lungs, from the size of a pea to that of a walnut. These foci are generally wedge-shaped, with the base on the pleural surface and the apex directed toward a larger bronchus. The foci are either sunken in and like meat (atelectasis) or more or less prominent, doughy, soft to tough, according to the duration of the process. They are reddish or grayish red, or even grayish yellow in color (worm-nodules, bronchopneumonia verminosa lobularis). On section these foci discharge a thick, reddish fluid which contains epithelia, pus corpuscles, ova, embryos and fragments of adult worms.

Not uncommonly extensive inflammatory areas are encountered, especially in certain species of animals (goats, sheep, calves and field hares), which are confined either to one or exceptionally to several lobes (bronchopneumonia verminosa lobaris sive diffusa). The affected portions of lung are like the spleen in color and consistency and they show on section dark red to black-red hemorrhagic spots, also dirty gray, crowded, even confluent spots. The expressed juice contains a large number of ova and worm embryos. In acute cases in calves the exudate often has a fibrinous character. In certain cases, the lungs contain vesicular nodules (as a rule, situated under the pleura) from the size of a millet seed to that of a hemp seed, which are found toward the apices and margins and which are of tough consistency (bronchopneumonia nodularis pseudotuberculosa s. alveolitis nodularis verminosa). These nodules are formed around an isolated, fully developed strongylus capillaris or a strongylus commutatus, or around aspirated ova. The nodules are light yellow to gray yellow in the presence of Str. capillaris, and red-brown to violet or blackish in the presence of Str. commutatus. After the death of the worms, the nodules undergo caseation and finally calcification.

In dogs pinhead-sized, translucent, pearl-like, exceptionally larger, bronchopneumonic foci are formed at the root of the lungs, sometimes also in other places; they are produced by the ova or embryos of Str. vasorum. In the cat the embolic transportation of embryos of Ollulanus tricuspis produces nodules similar to miliary tubercles with a hepatized pulmonary parenchyma in their neighborhood.

The bronchi contain an abundant mucoid or mucopurulent secretion, sometimes streaked with blood, in which the sexually mature worms are found. Under the microscope numerous ova and embryos are seen. One frequently sees the first worms at the end of the trachea, i. e., at its bifurcation, and these may completely obstruct the larger or the medium-sized bronchi, in dense masses, which sometimes contain only a few or no worms at all. The mucosa is swollen, reddened and sometimes streaked with hemorrhages or covered with papillæ—like pro-

liferations (*Braun*). Bronchiectases of varying sizes are rarely missed in the presence of worms.

Inflammation of the deeper layers of the bronchial wall can be recognized by its thickness, sometimes also by the presence of small purulent foci (peribronchitis). In such cases one finds a proliferation of pulmonary tissue around the bronchi, and white cicatricial bands are seen radiating from the bronchial wall into the air-containing tissue.

In hogs particularly pathologic changes are sometimes found only in the bronchi. These, however, frequently lead to atelectasis of the neighboring pulmonary parenchyma. The pleura pulmonalis of the affected animals frequently appears mottled, thickened, but rarely covered with fibrinous deposits, or adherent to the pleura costalis. According to Kitt, the Str. commutatus sometimes produces pleuritic abscesses.

Filaria tracheobronchialis produces in dogs wart-like nodules up to the size of a bean; they are arranged like mushrooms on the surface of the trachea and of the larger bronchi (tracheobronchitis verrucosa verminosa); these nodules contain worms which may project from under an uneven surface. In the pulmonary parenchyma there are many gray subpleural nodules, like granules of sand, and these contain each one worm. Osler also observed extensive bronchopneumonic inflammatory foci.

In severe cases the other organs present evidences of anemia and cachexia, especially subcutaneous edema and exudates into the serous cavities. A very intense gastritis is, according to Schlegel, of frequent occurrence. The bronchial glands are usually swollen.

Symptoms. The appearance of lungworm disease is possible at any time. It occurs, however, preferably in spring and fall or during the period of pasturing. The climax is generally reached in fall. According to Jerke, an outbreak in wild animals usually occurs in late winter or in spring when the resistance of the animals has been lowered by unfavorable food conditions. The same is true of wild hares. In domesticated animals, also, frequent outbreaks of the disease in late fall or at the beginning of winter feeding may have some relation to the changed feed conditions.

The reports of various authors on the appearance and occurrence of lungworm disease do not quite agree with each other. According to Railliet it is observed mostly during the warm season; according to Neumann, it occurs all the year around, especially between March and October; according to Zürn, in spring and in fall; according to Friedberger & Fröhner generally in fall; according to Dieckerhoff in calves toward the end of the pasturing season or even after its termination. Csokor states that lungworm disease in lambs appears in summer; in older animals only in fall. Moussu claims that the disease is prevalent all the year around, but reaches its climax late in the fall.

The period between the ingestion of the worm brood and the outbreak of the disease varies considerably according to the intensity of the invasion and to the individual power of resistance of the infected animal. Six to eight weeks elapse in

the majority of cases till the symptoms become manifest. After a minor invasion and in strong adult animals the symptoms of disease come on after several months, while after a severe invasion, symptoms may be noticeable after some days. Schlegel saw the disease 12 to 59 days after the infection of goats, while Kasparek saw, as already mentioned, bronchopneumonic foci in infected calves only 1½ to 8 days old.

In **sheep** and **goats** one first observes cough which in the beginning occurs only in few animals, after long intervals and preferably during exercise. The cough is short, dry and strong and spreads gradually to the greater portion of the herd. At the same time it becomes more frequent, weaker and tormenting. In severe cases one observes real paroxysms of cough. The bronchial secretion is expelled in large lumps through the mouth and in the former are found the worms, their ova and embryos; the latter by microscopic examination. The nasal secretion is usually seromucoid, but it does not usually contain embryos or ova. Sheep frequently rub their noses on the ground; sometimes with such intensity that a loss of substance occurs.

The respiration becomes more and more difficult, especially in sheep, so that the animals finally breathe convulsively. A variety of râles are heard over the thorax and trachea. Percussion usually shows nothing abnormal; but on percussing very attentively one occasionally finds dullness in circumscribed or even in more extensive places of the thorax, and sometimes also tympanic sounds. Bronchial breathing may likewise be heard. Ben Danon observed albuminuria. In extensive involvement of the lungs the temperature is elevated up to 40.5° to 41° C.

Emaciation and pallor of the mucosæ occurs in the further course of the disease, also a diminution in the elasticity of the skin. The disturbances in nutrition are often intensified by a complicating diarrhea. The embryos of Str. capillaris are always found in the feces (Schlegel). Finally edema appears on the entrance to the larynx, on the eyelids, the lips or on the whole front of the head; also on the lower thorax and the extremities. The animals become very weak and often fall down; when attempting to get up, the hind legs appear paralyzed and the patients succumb in complete prostration.

In **cattle,** especially in calves, one usually observes cough, at first strong and occurring at long intervals, but in the further course it becomes more frequent and more forced; when coughing the animals protrude the tongue from the mouth and expel masses of mucus which is sometimes mixed with blood and always coutains worms. In severe cases violent paroxysms with attacks of suffocation occur several times a day and one of them may lead to death by asphyxiation. The respiration is more or less accelerated from the start, later on forced. There may, however, be rapid variations in the respiratory disturbances, especially during the hot season (Hartenstein). Percus-

sion and auscultation show conditions on the whole similar to those observed in sheep. A febrile temperature exists only exceptionally (Scheibel).

After an intense invasion the symptoms may increase in severity so rapidly that the animals succumb within three to eight days. The clinical picture, however, develops slowly, as a rule; the cough becomes gradually weaker, the respiration more and more accelerated, the ingestion of food less. Emaciation, anemia and edematous swellings make their appearance.

In **dromedaries** and **camels**, lungworm disease is manifested in gradually increasing dyspnea, in serous and then mucous nasal discharge in which numerous embryos of the Str. filaria may be shown microscopically. The duration of the disease is two to four weeks, after which the animals recover or succumb (Piot-Bey).

In **hogs**, lungworm disease occasionally does not lead to disturbances of health. However, Str. paradoxus sometimes causes great losses, probably after an intense invasion, and the epizootic disease then takes a course similar to that in sheep (Sequenz, Czakó). In other cases the affected animals show only disturbances of nutrition.

In **horses** and **asses** the disease occurs exceptionally and only leads to a clinical picture, similar to that seen in verminous bronchitis of calves. Fatal cases have been observed repeatedly in asses (Steward).

Repeated attacks of respiratory difficulties are observed in **dogs** infected with Str. vasorum; these disturbances may disappear within a number of days or they may lead to the death of the animal. Ascites is developed in some cases. The disease occurs rarely, and generally only sporadically; however, an enzootic prevalence with numerous fatal cases has likewise been observed. Rabe observed obstinate cough and marked dyspnea in the presence of Fil. tracheo-bronchialis in the trachea and bronchi; Osler's Montreal cases occurred almost exclusively in young dogs and were characterized by fever, lack of appetite, weakness, paralysis of the hind extremities, convulsions, dry, short cough and vomiting. Railliet believes, however, that the bronchopneumonia found in these cases on post-mortem examination was due to distemper.

Spiroptera sanguinolenta, which occasionally may get into the respiratory passages, also produces chronic catarrh.

Lungworm disease of **cats** produced by the ova and embryos of Str. pusillus leads to frequent cough, often accompanied by vomiting, emaciation, diarrhea, cachexia and a fatal issue after two to three months. Broncho-pneumonia may occasionally be produced by the embryos of Ollulanus tricuspis. Müller & Neumann found Trichosoma aerophilum in the presence of catarrhal changes in the lungs of cats.

Lungworm disease is very rare in **rabbits** and in these animals leads to the same symptoms as in wild hares, where it frequently prevails to an epizootic extent, viz., accelerated and difficult respiration with frequent and dry cough and gradually increasing emaciation. The disease often ends fatally.

Course. The course varies very much, according to the in-

tensity of the invasion and the individual species of animals. The lungworms may frequently live in large numbers in hogs, but also not uncommonly in other animals, without disturbing the condition of health of the infected animals. Sheep and goats generally are affected most seriously after a more intense invasion, while the symptoms are milder in cattle and particularly in hogs. Among infected cattle, only weak calves will succumb, while hogs die from the disease only in very exceptional cases. The course of the affection is influenced by the occurrence of secondary infections.

The duration of the disease also varies a good deal; in *the great majority of grave, and hence unfavorable, cases, the* duration may be two, three or four months. Deviations in either direction are, however, frequent. On the other hand, the disease may last more than four months, even over one year, particularly in adnlt strong animals; while on the other hand, it may take a fatal issue within a few days or weeks (without exception only in younger animals, particularly in calves). In such cases one generally finds a larger bronchus obstructed by masses of worms, or a rapidly spreading bronchopneumonia. In its mild form the disease ends in recovery. In cases of medium intensity recovery occurs, usually in hogs, frequently in calves, after regulation of the diet; while sheep and goats, everything else being equal, generally succumb, especially younger, weakened animals, or those which are in the later stages of pregnancy. Even lambs that were originally puny, recover often in favorable weather to such a degree that they become fit for killing in winter (Miessner). The very severe forms *usually end fatally. The advent of improvement is marked by* a gradual diminution of the cough and by amelioration of the other symptoms. Recovery is not always complete. After the acute catarrhal symptoms have disappeared, some animals, especially sheep and goats, fail to improve in nutrition, a chronic condition of ill-health in time leads to death, or causes the animals to be killed.

Recovery is not always complete. Some animals, especially sheep and goats, still suffer in their nutrition after the disappearance of the catarrhal symptoms, and there may be a chronic wasting away with final death, unless the animal is slaughtered in time.

Diagnosis. Neither the symptoms of bronchial catarrh nor the subsequent cachexia are to be looked upon as characteristic symptoms of the disease. A diagnosis can only be made after the detection of the worms or their ova or embryos. The mature worms can be recognized with the naked eye in the sputum; if they are absent microscopic examination, the results of which are far more reliable than macroscopical inspection for mature worms, will detect the ova or embryos if present. Embryos of lungworms are also found in the feces of infected animals

(Schlegel, Piana, Eichhorn), also the eggs of Strong. paradoxus (Neveu-Lemaire).

Since cattle often swallow sputum which has been coughed up. Andersen recommends, after an attack of cough, the introduction into the pharynx of a rod armed at one end with a cotton plug; the secretion obtained on the plug of cotton is then examined. The feces are diluted with 1 to 3 parts of water, thoroughly mixed, and a drop placed upon a slide by means of a pipette (see page 340). In either case, all parts of the preparation must be systematically searched. Several preparations should be examined if necessary.

Symptoms which are more or less similar to those of lungworm disease are found in catarrhal pneumonia from other causes, as well as in enzootic pneumonia of young animals. These affections, however, lead from the start to more or less *marked disturbances of the general health with febrile elevation* of temperature and they do not lead to the formation of abundant masses of secretion; both forms of pneumonia usually occur in very young animals. Differential diagnosis between cases of lungworm disease associated with bronchopneumonia and other forms of pneumonia is only possible upon the detection of the worms and their broods. The disease is distinguished from pleuropneumonia by its usually afebrile course, by the absence of extensive areas of dullness and of pleurisy; further by the fact that cough remains for a longer time strong and convulsive. In young animals affected with pulmonary tuberculosis, the cough soon becomes weak. In Œstrus disease symptoms of involvement of the lungs are absent.

Prognosis. This depends upon the severity of the symptoms, also upon the age, nutrition and species of the affected animal. Everything else being equal, the prognosis is most unfavorable in the case of sheep and goats, most favorable in lungworm disease of hogs. Fatal cases are always more numerous among young than among adult animals. Sometimes, however, there may be no difference in this respect (especially in infection with Str. capillaris or Str. commutatus). In lungworm disease of sheep the mortality varies from 10% to 70%.

Treatment. Inhalations of irritating and disinfecting vapors are of very doubtful value, even at the commencement of the disease; at best they may stimulate cough and thereby promote the removal of the worms and of their brood from the air passages. In order to apply them, the animals are made to inhale smoke, generated in closed places by burning tar, creolin, horn-substances, stinking animal oil, or the vapors produced by heated oil of turpentine.

The most serviceable procedure seems to be the intratracheal injection or vaporization of liquids capable of killing the parasites. Even this method is fully successful only in cases in which the lung tissue is not deeply involved; on the other hand, the physical nature and the mode of application of the

antiparasitic liquids are of essential consequence. Oily mixtures have the advantage that they remain in the air passages longer than aqueous solutions without being absorbed; but drugs dissolved in oil act less rapidly and less energetically; they do not mix with the phlegm of the respiratory passages and therefore cannot reach the worms contained in it. Both oily mixtures and aqueous solutions follow the law of gravity and reach largely or wholly only the anterior lung portions, so that the other parts of the lungs are not influenced. Fluids which are injected into the trachea in a fine spray, are aspirated into the bronchi, with the inspiratory air; it must be taken into consideration, however, that no air current moves in the lung portions which are occluded by exudate or by bunches of worms, or in their bronchi, and that here the aspiration of sprayed fluids cannot be effective therefore. The combined application of aqeous and oily solutions and of sprayed liquids seems to be worthy of recommendation.

Of the oily mixtures the following may be mentioned: Ol. terebinthinae rectific. Ol. Olivae (Ol. lini, Ol. rapae) āā 100.0 Creolini purissimi 10.0. Of this mixture sheep receive 5 gm. per head; calves 15-20 gm. per head in single applications on three successive days. Vaeth's mixture is as follows: Ol. caryophyllorum, Ol. terebinth. āā 100.0 Acidi carbol., Ol. cadini āā 2.0; 10 gm. for calves in one application. Schafnicky's mixture: Ol. oliv., Ol. terebinth., Ol. cornu cervi āā 0.5 gm. Of this 0.5-1.0 c. c. daily for sheep; to be continued for 1 or 2 weeks. De Mia's mixture: paraffini liquidi, Ol. terebinth. āā 100.0, Ol. caryophyll., Acid. carbol. āā 2.0, for calves 10 c. c. four days in succession. Elvire's mixture: Ol. terebinth., Ol. papaver āā 100.0, Acid. carbol. 2.0, for sheep 10 c. c. three days in succession. Neimann's mixture: Sol. Lugoli (1:10:100), Ol. terebinth., Ol. oliv. āā 0.5-0.8; for sheep the whole dose, to be repeated the next day. Tapken's mixture: Sol. Lugoli (1:10:100), Ol. terebinth. āā 10.0, Ol. oliv. 20.0; for cattle 8-10 c. c.—Creolini angliee 10.0, Ol. terebinth., Ol. oliv. āā 100.0; for sheep 5 c. c., for calves 15-20 c. c., in each case three days in succession. Bergeon's mixture: Creosoti 20.0, Ol. amygdal. 100.0; for calves 5 c. c. in one dose, after a pause of four days give 20 c. c. for several days. The following aqueous solutions may be mentioned: 1% carbolic acid solution (lambs 5 c. c.; calves 20 c. c. in single applications, several days in succession). Wessel & Vaeth found this effective in calves, Kroening in lambs. A 1% solution of creosote (1 part creosote āā 50 parts of rectified spirits and dist. water) is recommended by Scheibel for tracheal spray in cattle. Of 0.1-2% solution of Potass. picronitrat., 5 c. c. for sheep (Peters), 20-60 c. c. for calves, according to age (Nielsen). In dromedaries Piot-Bey employed Tinct. Op. 0.5, Ol. terebinth., Ether sulph. āā 10.0, Sol. Acid. carbol. in aq. (1:100); 20 gm. for each application.

According to Hermes and Freeborn and Ransom and Hall, good results were achieved in America in sheep, goats, calves and hogs by an instillation of chloroform into the nostrils (sheep and goats 1.5 cc., calves 6.5 cc., into each nostril). The nostrils are then closed either with the hands or with a pledget of cotton until the animals show signs of be-

coming anesthetized. They are then placed in a lot, free from vegetation, and two hours later are given a dose of Epsom salt. The treatment is repeated three times, at the most, at intervals of 3 to 5 days. The worms, larvæ and eggs are said to be ejected through the coughing produced by the chloroform.

Tracheal injections are given to large animals standing; to smaller ones lying on the side, the head being held on the stretch. The Dieckerhoff syringe with a tracheal trochar is better than a simple hypodermic syringe. Before the needle or the tracheal trochar is withdrawn from the trachea, some pure water should be injected in order to prevent part of the drugs from lodging in the injection-wound, where they might cause painful swellings.

Scheibel uses in the treatment of cattle a modified spraying apparatus which consists of an elliptically bent tracheal tube 0.4 cm. wide, with a shield and two eyes, so that the apparatus may be fastened with strings to the neck. The tube is armed with a trochar, so that it can be pushed into the trachea of the animal. The trochar is withdrawn and a dichotomously divided canula is then inserted into the tube. Creosote solution is then sprayed through the canula into the trachea and bronchi, just as is done when a Frick spray apparatus is used; this is to be repeated for one or more days, at least 8 minutes each time. The vessel containing the solution must be held lower than the canula, otherwise the fluid would simply run in without being finely divided (Zwaenopoel and Coppens). The spray must be interrupted if an attack of cough sets in. The tube should be left in the trachea during the whole period in which this treatment is employed.

On account of the sensitiveness of the larvæ to copper salts, von Linden recommends copper salt licks, either in the form of granules or in blocks (the latter best for game). Figured on the basis of pure copper, the preparation should be made up of a mixture containing 1% copper chloride or copper sulphate with table salt (the copper salt should be free from arsenic, the table salt free from iron). It is administered for 4 to 6 weeks. Mature cattle received daily doses of 10 grams, calves, sheep and goats 5 grams, mixed with the grain or dissolved in a liter of water. Lambs and pigs receive doses of 1.5 grams, either mixed with grain or dissolved in one-fourth to one-half liter of milk, etc. In case of the presence of a severe diarrhea, the less irritating cuprocarb (Kupferkohle) should be given by the tablespoonsful or teaspoonful. Intramuscular or intravenous injections of 10 to 15 cc. of a 5% solution of dimethyl-glycocoll-copper or a 5% colloidal solution of copper silicate, given every two weeks, are also recommended. The latter is injected alternately on the right and left side of the neck or in the region of the croup (on the average 8 to 10 injections in all). The salt lick treatment is said to be more reliable as compared with the injections; in particular, very weak animals can still be saved by it. Concerning the curative effect of the treatment, Miller was not able to reach a definite conclusion in his cases with calves, but following the intramuscular and subcutaneous injections he observed extensive swellings. The effectiveness of the copper salt licks and of copper in general is denied by Olt, Gmeiner, Jerke, Knuth and others while von Kries found them to be reliable.

For the destruction of the brood which is ingested with feed or water and remains in the stomach or intestine for a time, the internal administration of vermifuge remedies is suitable as long as there is danger of infection. For sheep 0.2% solution Potass. picronitrat. in 45 c. c. of oatmeal gruel (Nielsen); for calves Acid carbol., Ol. animal. foetid., aa 3.0, Ol. terebinth., Ol. oliv. aa 100.0, tablespoon doses with gruel made from linseed (Schoettler); for lambs, 1% solution of Cu. bichlorid. (5 c. c. daily) in milk (v. Linden). The remedies employed for the treatment of stomach-worm disease may be employed also. v. Linden recommends salt licks or licking stones (bichloride of copper and common salt 1:100); these and other licking stones may be used in treating wild ruminants which may also be given the copper bichloride (0.05 gm. per dose) mixed with the feed. The effectiveness of licking stones is frequently denied.

Stimulation and strengthening of the animals are of the utmost importance because experience has shown that strong animals sometimes survive even a severe attack. The animals should therefore have nutritious food, if possible cereals to which have been added bitters and preparations of iron. Pasturing animals should also receive nutritious food and hay. In order to prevent repeated invasions the pasture might be changed or the sick animals might be stabled. Animals which are very sick and which do not improve in spite of proper treatment should be slaughtered.

Prophylaxis. Lowly situated, marshy and damp pastures should be avoided, also ditches the borders of which are grown with reeds. Care is particularly necessary in continued rainy weather, and young animals which are especially susceptible must be protected. The drainage of low pastures and the extinction of the worm-brood by scattering superphosphates or Thomas-phosphate over pastures and feeding places may be taken into consideration. These substances are scattered in double the quantities used ordinarily for fertilizing. In such neighborhoods where the disease has become permanently endemic, dry feeding offers the best protection (Schlegel). Preservatives which are praised as effective are useless, but one might water the animals before they are driven to the pastures, because this might cause them to avoid drinking water from pools and marshes. The sputum coughed up by the sick animals, the feces and the bedding-straw, the respiratory tract of slaughtered animals or those dying spontaneously must be destroyed. In a herd which is already infected, the young animals should be separated from the adults. According to von Linden, a biweekly treatment with copper salts, as a rule, protects the animals against infestation.

5. Animal Parasites in the Air Passages of Fowl.

(a) Syngamus. (Gapes.)

Historical. A disease caused in chickens and turkeys by Syngamus trachealis was first observed in 1779 by Wiesenthal in Baltimore. It has since been reported repeatedly and has been studied more carefully by a number of observers (Leuckart, Ehlers, Railliet, Mégnin, Walker).

Occurrence. The disease which is produced by syngami appears to be especially prevalent in America, England, Italy, France and Germany, and it prevails preferably in enzootic or epizootic distribution among pheasants bred in larger numbers in captivity. Syngamus trachealis invades especially pheasants, chickens, turkeys and peacocks, while Syngamus bronchialis *infects water fowl (geese, ducks); however, S. trachealis occasionally* affects other domestic birds, especially pigeons (Tossi) and room birds kept in cages. Syngami have also been found in partridges, American blue-jays, in cardinals, magpies, rooks, swallows, wood-peckers, storks, starlings and ravens. Young birds are generally affected.

Wiesenthal estimated that the loss by syngami in the invaded parts of the United States amounts to 80% of all young chickens; Crisp states that England loses annually about one-half million of chicks. In a large pheasant breeding establishment of France there was a daily loss of 1,200 pheasants. Klee estimates the loss in Germany to several hundred pheasants annually. Syngamus laryngeus occurs in the larynx of cattle in the southern parts of Annam, but does not lead to any trouble.

Etiology. Of the genus Syngamus, belonging to the family Strongylidæ, the air passages of birds are invaded by Syngamus trachealis Siebold (Strongylus trachealis, S. primitivus) and Syngamus bronchialis Mühlig.

Syngamus is a slender, red cylindrical worm. The head contains a mouth-opening surrounded by a strong chitinous ring. The posterior end of the male, which is 0.2 to 0.6 cm. long, terminates in a ribbed bursa which covers two spicula. The female is three or four times as long. The oval ova (Fig. 116) are 85 μ long, provided with a double shell. The latter contains at either end a roundish opening closed by a delicate membrane. They contain a segmented mass or a developed embryo. In the trachea of fowl the male and female are usually found in copulation; the two look like one worm, branched under an acute angle.
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Fig. 116. Egg of Syngamus trachealis, from the droppings of a hen.

Evolution of the Syngami. The vulva of the female being closed by the male which is attached to it, the eggs are liberated only after the mother animal is torn or has rotted, in the air passages, or after being expectorated. Railliet claims that the eggs can be discharged from the vulva in spite of the permanent copulation. When the eggs get on moist substances, into water or into the air passages, the eel-shaped

embryos develop, according to the temperature, in seven to forty days; if they are taken up by suitable hosts, they are arrested, according to Walker, in the walls of the gullet or of the crop, or they migrate there from the stomach; they perforate the wall of the gullet and finally reach the lung and the air passages. It is possible that a transportation through the circulation takes place also, in like manner as in the lungworm disease of mammalians. The larvæ mature in the air passages of the host and then copulate. Ehler's observation that the disease may be produced artificially by feeding eggs that contain embryos, speaks in favor of a direct evolution without an intermediary host.

Infection takes place by embryos or larvæ of Syngamus being ingested with food or drinking water, into which the embryos have come with the expectoration or with the feces of the host. Infection may occur by swallowing rainworms, which is suggested by the observation of Walker that in infected regions rainworms may contain larvæ of Syngamus. There is also a possibility of direct infection by contact with diseased animals if their discharges are picked up by healthy birds Even an autoinfection through embryos that have been liberated in the air-passages cannot be denied, in view of the results of an experiment made by Walker. This is confirmed by Rossi's observation that young pigeons were infected by being fed by their diseased mother birds; the young pigeons died of a verminous inflammation of the crop.

The disease is introduced into healthy flocks by infected domestic birds, also by wild birds, crows, starlings, which frequently harbor Syngamus without being manifestly ill (Railliet, Klee). Klee found syngami in one-half of the crows caught in the neighborhood of an infected pheasant establishment.

Anatomical Changes. The worms are generally found in large numbers (30-40) in the air passages; Syngamus trachealis preferably in the trachea, Syngamus bronchialis deeper in the bronchi. The mucosa to which the worms adhere shows catarrhal changes, also occasionally small abscesses; the lungs are frequently affected in Syngamus bronchialis infections. Both species are not uncommonly found in the air vesicles.

Symptoms. The sick birds begin to cough and they shake their heads repeatedly; they expel masses of tenacious mucus from the mouth. They often open their bills (hence the common English name of the disease "gapes") and have a whistling breath. Emaciation soon develops in spite of a good appetite. The respiration becomes more and more difficult, abundant mucus collects in the buccal cavity, the appetite gradually disappears, the birds stand with ruffled feathers and they die totally exhausted or even earlier during an attack of dyspnea. Older birds sometimes recover if only few worms are present.

Diagnosis. The disease can be diagnosticated upon detection of the worms or ova coughed up with the secretion or discharged with the

feces. The worms may be seen in the trachea of larger birds on intense illumination of the larynx, if this is pushed upward toward the buccal cavity with the trachea. In smaller birds they become visible, if after removal of the feathers, the trachea is drawn out with the skin covering it and held before a strong light (Renné, Lichtenstern).

Treatment. The worms which are situated in the upper portion of the trachea can sometimes be extracted by the aid of a long slender pair of forceps. For the destruction and removal of the worm, Kaupp recommends the introduction into the trachea of a horse hair or feather steeped in oil of turpentine. Voitellier recommends the inhalation of acidum sulfurosum (in water, 1:400) or phenol, claiming 95% cures. Verwee used inhalation of the fumes of oil of turpentine combined with sodium salicylate in 1:1000 solution as drinking water. Good results have recently been obtained by the intratracheal injection of a 5% solution of salicylate of sodium (Klee).

1.0 cc. of this solution is introduced with a syringe provided with an obtuse, bent needle. It is injected from the pharynx into the larynx or trachea. One may also push the needle into the trachea, which is held between two fingers. Since the trachea of smaller birds is hard to enter in this manner, it is sometimes necessary to lay it free through an incision into the overlying skin. The parasites drop off after the injection and are expelled by violent attacks of cough. Lichtenstern did not succeed with intratracheal injections of pieronitrate of potassium.

Prophylaxis. The expelled tracheal secretion, the feces and the cadavers of diseased birds must be destroyed; the floor and the poles of the coops must be disinfected; pools in the barnyard must be dried out; the feeding and drinking vessels must be cleaned repeatedly. Mégnin advises to spread denatured common salt on the floor (250 gm. to each 100 square meters of floor space), or sprinkling the floor with 0.1% solution of sulphuric acid, also drinking water containing 1.3% of salicylate of sodium. Sprinkling the soil with burnt lime may likewise be serviceable. Sick birds must be separated from healthy ones. The destruction of crows and other birds carrying the infection and the collection and destruction of earthworms are other means of combating the disease.

(b) The Air-Sac Mite of Chickens. Cytodites nudus.

Cytoides nudus (Cytoleichus sarcoptoides), a mite, 1½ mm. long, the female of which is oviparous, occurs occasionally in large numbers in all parts of the air passages, especially in the large air cells. The evolution of this mite is unknown. It occurs preferably in the air passages and larger air cells of chickens and pheasants, where it forms very small white, occasionally motile, points visible to the naked eye, which form a hoar-like deposit when much crowded together. Exceptionally the mites are also found in the air spaces of the bones; and the mucosa of the bronchi rarely becomes catarrhal under their

influence. Then the mites are found in an abundant mucopurulent mass or in croupous membranes.

Cytoleichus sarcoptoides frequently does not produce any symptoms of disease, even if present in large numbers. In other cases it causes a catarrhal condition of the air passages with cough which, according to Zürn, is accompanied by a peculiar sound, as if a small foreign body had gotten into the trachea. In an exceptionally extensive invasion there may be severe bronchitis with the formation of so much secretion that death may occur from suffocation (Mégnin). Rosencrantz (B. t. W., 1909, 757) saw numerous fatal cases exclusively among cachectic adult chickens in whose peritoneal cavity there were innumerable sarcopteslike mites; inflammatory changes were however absent.

The disease cannot be diagnosticated from its clinical symptoms as long as the birds are alive. An effective treatment is unknown. Inhalations of tar or iodine vapors might sometimes be tried.

Laminosioptes cysticola (Symplektoptes cysticola). Kasparek saw an enzootic among pigeons caused by symplektoptes cysticola. In distinction to previous references, according to which symplektoptes lives only in the subcutaneous tissue, it caused in Kasparek's cases an inflammation of the air passages, progressive inflammation and finally death of the animals. The mite-like parasites were found in the air passages; also yellowish, whitish, elongated nodules in the lungs, each containing one parasite.

Monostomum flavum. (Typhlocoelum obovale.) This parasite belongs to the Trematodes; it is of ochre color, 1.2-1.5 cm. long. Its eggs, 154-180 μ long, are brownish yellow. The parasite appears in the air passages of ducks in Brazil and causes attacks of suffocation, because of their great numbers, to which the animals succumb. The evolution of the parasite is similar to that of the liver-fluke; the larvæ develop in a snail, Planorbis corneus (Neveu-Lemaire, Parasitologie des animaux dom. Paris, 1912).

SECTION IV.

DISEASES OF THE LUNGS.

1. Congestion of the Lungs and Edema of the Lungs. Hyperaemia et Oedema pulmonum.

Hyperemia of the lungs consists in an abnormally high amount of blood in the pulmonary capillaries, which may be due to an increased inflow of blood (hyperaemia pulmonum activa) or the condition may be brought about because, for some reason or other, the blood cannot flow without impediment from the lungs toward the left side of the heart (hyperaemia pulmonum passiva). Edema of the lungs occurs in consequence of an extravasation of serous fluid into the alveoli and bronchioles and also into the interalveolar tissue.

Etiology. Hard labor may produce an increased supply of blood to the lungs, since the heart which works energetically and rapidly may bring an increased amount of blood into the pulmonary artery. Active hyperemia on this basis, therefore, frequently occurs in racers, in hunters and army horses, and in hunting dogs, especially in summer when the warm air also has a weakening effect upon the pulmonary vessels. In a considerable proportion of such cases we are, however, rather dealing with heat-stroke or with acute cardiac weakness due to passive hyperemia (for details about the disease called "summer foundering" by Bongartz, see in the chapter on heat-stroke).

The disease under discussion appears in horses and, perhaps, also in other animals in combination with rheumatic affection of the intercostal muscles, during long railroad transportation, particularly if the animals are crowded in the car (Dieckerhoff, Sigl). Overheating of the body, exertion and excitement are probably the injurious factors (Dieckerhoff describes this condition as a disease sui generis under the name of Pleurodyuia).

Very hot air or air mixed with acrid and irritating vapors may produce au increased blood supply by dilating the pulmonary vessels or by acting as an inflammatory stimulus; this has been shown by observations made after conflagrations.

Hyperemia seen in the early stages of acute inflammatory processes also belongs to this group.

A collateral hyperemia is produced when the blood current in the lungs meets with a rapidly forming extensive impediment such as occurs in pneumothorax on the healthy side and in considerable elevation of intra-abdominal pressure on both sides.

Congestive hyperemia is one of the usual concomitant conditions in stenosis of the left venous ostium or in mitral insufficiency. In the majority of cases, however, it develops in consequence of cardiac weakness in dilatation of the heart, in non-compensated valvular disease of the heart in consequence of myocardial degeneration in many acute infectious diseases. Compression of the pulmonary veins in pericarditis, decrease of the negative intrathoracic pressure in bloating of the stomach or of the intestines may be the cause of congestion of the lungs. The prolonged recumbent position on one side may also cause hyperemia in the lower portions of the lungs without the contribution of the above factors (hyperaemia hypostatica), because in such a position the venous blood has to overcome the effect of gravity.

The pathological significance of hyperemia of the lungs rests upon the production of a certain rigidity of the lung tissue combined with decreased elasticity of the same, due to rigidity of the engorged capillaries as a result of increased tension of their walls. Furthermore, a decrease in the lumen of the alveoli and the small bronchi also takes place because the dilated capillaries project into them. In case of passive congestion, a decrease in the rapidity of flow of the blood leads to accelerated breathing, either directly by the overloading of the blood with carbon dioxide or by reflex action, and in the latter case probably before an overloading of the blood with carbon dioxide has taken place.

Edema of the lungs is also due to various causes. All those factors which produce hyperemia of the lungs may also produce edema of the lungs if they are acting more intensely or over a long period of time. Those types of edema which are due to alterations of the vessel walls brought about by irritating substances are easily explained, for in these cases the extravasation of blood serum is a sequel of disease of the vessel walls as is generally the case in inflammations. A similar mode of origin of pulmonary edema is that seen in association with acute pneumonia and in the course of some infectious diseases (anthrax, malignant edema and other septicemic affections). Bacteria and their toxins then need not necessarily act directly upon the pulmonary tissue, but their inflammatory action may be exerted through the blood circulation. A similar effect may be manifested by certain vegetable or mineral poisons or by decomposition products of the organism; intense edema

of the lungs which is sometimes seen in severe disease of the kidneys is probably of a similar origin.

In the majority of cases the edema is due to stasis of the blood. It frequently develops on this basis shortly before death from cardiac weakness, combined with insufficient expansions of the lungs. Valvular lesions in the left venous opening often lead to edema of the lungs even when compensated, because the blood cannot flow freely from the veins of the right side of the heart even when the heart muscle acts well. Cardiac weakness exclusively of the left side is very rare as long as the right side contracts powerfully (Krehl). Hypostatic hyperemia may also lead to edema of the lungs.

Nutritive disturbances of the vessel walls in cachetic conditions likewise predispose to edema of the lungs.

The pathological significance of edema of the lungs, besides having a deleterious influence upon the respiration, resting upon the same basis as hyperemia of the lungs, consists in the occlusion of the alveoli and air passages by the edematous fluid, thus bringing about a high degree of disturbance in the gas exchange in the lungs.

Anatomical Changes. The consistency of the lungs is somewhat increased in pulmonary hyperemia and they are possibly also somewhat bloated, the cut surface is darker and discharges blood freely. If the congestion has lasted a long time the lungs become tough in consequence of an increase of connective tissue and the cut surface appears rust-brown owing to the presence of haematoidin granules (so-called induratio brunea). Hypostatic hyperemia is usually found only on one side or on part of one side; here the pulmonary parenchyma is of spleen-like consistency because the alveoli are filled with blood corpuscles and blood plasma. This condition is called splenization or, wrongly, pneumonia hypostatica.

Pulmonary edema is recognized by a fine foamy fluid, mixed more or less with blood, which oozes out in large amount from the cut surface of the lung which has become less elastic. The bronchi are filled by a similar fluid and the air contents is very much diminished. One also observes the usual signs of death from suffocation.

Symptoms. The external symptoms of pulmonary hyperemia and of edema are on the whole very similar and consist in the signs of rapidly increasing dyspnea and attacks of suffocation. The mucosæ of the head become cyanotic, the eyes protrude and the jugular veins swell up. The respiration is much accelerated and forced and soon becomes rattling. In edema of the lungs the much dilated nostrils discharge a foamy, serous or serohemorrhagic fluid. A short dull cough is heard periodically. The percussion sound in hypostatic hyperemia is dull over the lower portions; otherwise it is normal or may be

somewhat dull and higher in pitch on account of the increased tension of the intercostal spaces; in severe cases of pulmonary edema it is likewise dull, possibly here and there tympanitic. Vesicular breathing is lessened in hyperemia, or on the contrary, rough; in edema it is much weakened, sometimes entirely absent; crepitant and rattling rales may be heard. The heart beat becomes *pounding*, the accelerated *pulse* is *at first tense* and full, but later, or in concomitant heart disease from the start, small and weak.

All these symptoms may disappear as rapidly as they came on, and the animals may completely recover within 6 to 12 hours, especially in active hyperemia, while in other cases suffocation comes on with the increase of the symptoms within an equally short space of time (apoplexia pulmonum). On the other hand congestive hyperemia usually develops gradually and, even in severe cases, leads to death only after several days. Pulmonary edema also causes, at times, death very rapidly and almost in an apoplectic manner (apoplexia pulmonum serosa).

In pleurodynia, respiratory disturbances, tenderness to pressure of the intercostal spaces and acute bloating of the lungs are observed, all symptoms being due to the tenderness of the intercostal spaces. These symptoms usually disappear in four to six days without leaving a trace, sometimes, however, a fibrinous or serofibrinous pleurisy develops subsequently, which may cause the death of the patient.

In *shipping fever of horses*, *Forgeot* observed depressed appetite, fever, icteric or brick-red discoloration of the conjunctiva, labored breathing with a normal percussion and auscultation sound or, at most, a decrease in the vesicular respiratory sounds in the lower regions of the lungs, weakness in the hind quarters, dark discoloration of the urine, a mucous coating of the fecal balls, and light constipation.

Diagnosis. Both hyperemia and edema of the lungs can usually be diagnosticated easily if one considers the usually sudden onset of the disease with the symptoms described and if a history or the signs of the external irritants mentioned or of an existing primary affection are obtained. In heat-stroke not only dyspnea is observed, but also marked cerebral depression, physical weakness and a considerable elevation of temperature. In septic infectious diseases (anthrax, swine erysipelas) the symptoms of a severe febrile general affection are present from the start, coming on without any discernible external influences. Edema of the lungs is distinguished from diffuse bronchitis by the absence of fever and by the presence of crepitant râles and a foamy discharge from the nostrils. In pulmonary hemorrhage foamy blood is discharged from the nostrils.

The **prognosis** is usually not unfavorable in active hypere-

mia occurring in otherwise healthy animals, if proper treatment is at once instituted. Passive congestive hyperemia, hypostatic hyperemia and edema of the lungs frequently terminate fatally. Symptoms of cardiac weakness are of unfavorable prognostic significance as is also elevation of temperature.

Treatment. Rest is most important for the patient. Extensive blood-letting considerably relieves the heart and diminishes the blood pressure in the lungs, so that they are again able to expand properly; blood-letting, carried out at the proper time, may save the life of an animal in acute pulmonary hyperemia; but even in congestion in the lesser circulation it is advisable to withdraw a moderate amount of blood.

Subcutaneous injections of excitants (camphor, caffeine, ether), rubbing or irrigation with cold water are especially indicated if the pulse is weak. Cardiacs are serviceable in cardiac disease. As long as the pulse is sufficiently strong, one may try in edema of the lung subcutaneous injections of atropine (0.03-0.05 gm. for large, and 0.005-0.01 gm. for small animals), scopolamine (0.01-0.05 and 0.003-0.01 gm.).

By way of prevention, animals with cardiac disease should be protected against hard work, rapid running, etc.; their evacuations should be regulated. Sick animals which are down must be turned frequently.

Gas Poisoning in Battle. During the past World War, poisoning by gases in horses, dogs, and homing pigeons was observed during gas attacks. These were at first reported as so-called irritating gases with only an irritating effect upon the mucous membrane (brominated methyl-ethyl-ketone). Soon, however, poisonous gases were used (chlorin, phosphorus, chloral-picrin, chlorinated acetone, carbonic chloride, phosgene, stannic tetrachloride, diphenylchlorazon, trichlormethylchloroformiate, diethylsulphide. Of these, chlorin is still fatal in a concentration of 50 mg. per cubic meter and phosgene in a concentration of 7 mg. per cubic meter and others in still smaller quantities. The generally observed greater resistance of horses as compared with man, dogs, and homing pigeons can be explained by the fact that the horse breathes the gases in a lesser concentration because its nose is more removed from the ground than that of the men or smaller animals in the trenches or lying on the open ground. The effect of the gases is probably also reduced while passing through the relatively long nose of the horse (Schulze and Otto).

These gases produce effects similar to those of gases inhaled during conflagrations; however, the effect of the different gases varies with the concentration and the length of time during which they are inhaled. Their harmful effect is especially directed against the epithelium of the capillaries of

the alveoli and bronchioles (inflammatory edema); however, the other regions of the respiratory tract and the pharynx as well as the conjunctiva are also affected, while some of them even affect the skin (catarrh, penetrating inflammation, necrosis of the skin). Following absorption, general symptoms of poisoning may be produced.

Usually the inhalation of the gases is immediately followed by severe coughing, which, in turn, is followed, sometimes in the course of the next 24 hours, by symptoms of bronchitis and edema of the lungs, such as profuse grayish-white or greenish-yellow, sometimes bloody, foamy, discharge from the nostrils, severe dyspnea, especially upon inspiration with trumpet-like dilatation of the nostrils and a staring and frightened look. Furthermore, crackling, rattling, and often purring or whistling sounds are observed over the thorax; later a dullness upon percussion and decreased vesicular breathing due to the intense edema or bronchial pneumonia. Further symptoms are a slowly mounting fever, a rapidly increasing weakness and acceleration of the pulse (70 to 90 per minute), a clear amber-colored discharge from the eyes, light swelling of the eyelids, sometimes *disturbances in deglutition*, rigid position of the body, apathy, lack of appetite, and lack of thirst. Pleuritis frequently follows later.

Following the action of some gases with a distinct drugstore-like or garlic-like odor, all phenomena of irritation are lacking during the first few hours. Only after about 4 to 6 hours, one recognizes a gradually increasing swelling of the eyelids and the usual symptoms of a severe conjunctivitis, opaqueness of the cornea, discharge from the nostrils, frequently a rise in body temperature and an increase in the pulse rate, without the lung tissue being simultaneously affected. After 5 to 7 days necrosis of the skin penetrating to a varying depth is observed. Recovery finally takes place (Schock).

Course. Some animals die soon or within a few hours from suffocation or paralysis of the heart even before severe disturbances of the respiratory organs have had time to develop. Cases with plainly manifest symptoms of edema of the lungs and bronchitis usually terminate fatally within 48 hours. In cases with a less rapid course, particularly in about 30% of the cases (Beck and Tiljak), improvement takes place beginning about the fourth or fifth day and the animals finally recover; however, a shortness of breath (bronchitis) can be observed for a long time thereafter. Bronchopneumonia which usually terminates fatally develops in about 10% of the cases. In case the symptoms of intoxication appear rather tardily, the animals usually recover in 3 to 4 days.

Treatment. The animals should be carefully led out of the gassed territory and placed in a well-ventilated stall or in an

open corral. Complete rest is necessary. A large quantity of blood should be drawn from the jugular vein and at the same time a 1 to 2% solution of calcium chloride (500 cc. three times daily) injected intravenously. Furthermore, inhalation of oxygen or intravenous injection of ozonal, cardiacs or atropin may be used. When the appetite returns, succulent feed or at least roughage free from dust should be provided.

Prophylaxis. Gas masks containing various absorptive substances (sodium superoxide, sodium phenolate for chlorin gas; hexamethylinetetramin for phosgene) should be applied to horses and dogs. These masks are furnished in three different sizes for horses and are only placed around the upper jaw, leaving the lower jaw free (thus preventing breathing through the nose). In cases where no gas masks were available, feed bags containing moist hay or feed bags moistened with sodium thiosulphate and potash were placed upon the animals with varying success. Furthermore, covers soaked in salt water were placed over the heads of horses and all movements of the horses prevented during gas attacks. For homing pigeons, the cages were similarly protected by masks.

2. Pulmonary Hemorrhage. Haemoptoe.

(*Blutsturz* [German].)

As pulmonary hemorrhages all those cases are designated clinically where the source of the blood is found in the air passages behind the larynx or in the lungs themselves; it is not possible to differentiate clinically hemorrhages arising from the bronchi or from the pulmonary parenchyma.

Etiology. Haemoptoe often is a consequence of hyperemia of the lungs (see page 618). Pulmonary hemorrhage is, therefore, seen preferably in horses after overheating in rapid running or in consequence of hard work, also as part of the clinical picture of heart disease. Guittard often saw pulmonary hemorrhage in young, well-nourished chickens after taking cold during rough weather.

Hemorrhages from the lungs, which occur frequently in the course of infectious diseases, can be explained by a diminished resistance of the vessel walls. Diseases of this kind are, particularly, purpura haemorrhagica, smallpox, anthrax, septicemia, pyroplasmosis in horses, acute pneumonia, etc. Deficient nutrition of the vessel walls undoubtedly is a factor in such affections as leukemia and renal diseases and other diseases leading to cachexia. Frequent hemorrhages in hemophilia likewise depend upon a diminished tissue resistance.

Necrotic processes in the mucosa of the air passages and in the pulmonary parenchyma may produce hemorrhages by

erosion of vessels. This occurs in glanders in horses and in tuberculosis in cattle, in pulmonary gangrene, in ulcerating neoplasms, and in cavity formation in the lungs which occurs sometimes after pneumonia (Fröhner, authors' own observation).

Aneurysms of the aorta or of the pulmonary artery which happen to be adherent to a bronchus may rupture and produce a fatal hemorrhage. Thrombosis or embolism of the pulmonary vessels may likewise cause minor hemorrhages and this occurs often in valvular disease of the heart. A rare cause of haemoptoe are injuries to the lungs.

Symptoms. Hemorrhage of the lungs does not always become manifest because small amounts of blood may remain an the air passages and may be absorbed rapidly or they may coagulate and subsequently disintegrate. An insignificant hemorrhage manifests itself sometimes in such a manner that an existing nasal secretion or bronchial sputum contains small bloody dots or streaks, or that it looks saffron-yellow or rust-brown in consequence of an intimate admixture of blood. (A saffron-yellow color is seen particularly in pneumonia, a rust-brown color in valvular disease of the heart.)

Severe pulmonary hemorrhages lead sometimes to a frightful flow of blood out of the nostrils and mouth, and intense dyspnea is associated with such an occurrence. The flow is usually bright red and foamy and small air bubbles may even be seen in a coagulum which may have formed. The respiration is much accelerated and convulsively difficult; the animal betrays great anxiety, trembles, perspires, staggers and finally falls down if the hemorrhage continues. The mucosa, at first cyanotic, becomes gradually paler; the pulse becomes accelerated and filiform. Râles are occasionally heard over the lungs; also over the trachea; sometimes crepitant sounds are audible. Finally the pulse can no longer be felt, the peripheral parts of the body become cold, the animals fall down and die in convulsions.

Diagnosis. Hemorrhage of the lungs may be recognized from the bright red and foamy character of the blood voided. The source of the hemorrhage however is recognized only with difficulty. It may occur that blood coming from the posterior parts of the nose, as well as blood coming from the mouth, pharynx or esophagus flows backward to be then expelled with attacks of coughing. Under these circumstances the blood does not contain numerous air bubbles and the examination of the parts named and of the thorax will usually afford information about the source of the blood. In hematemesis the blood is expelled with vomitory movements, it appears dark in color, and is often mixed with coagula; it reacts acid and frequently contains particles of food. One must, however, not forget that

the act of vomiting is brought about in smaller animals by coughing, that vomited blood may subsequently get into the air passages, that blood from the lungs likewise becomes dark later on and that a large amount of blood will remain alkaline even in the stomach. Hence, in making a diagnosis, it is necessary to consider all circumstances and the condition of all internal organs.

Prognosis. Usually the animals appear in good condition after inconsiderable hemorrhages, larger animals even after a loss of 3-4 kilograms; at most they show a transitory weakness. After a large loss of blood, more or less anemia persists for some time and may be followed by cachexia and death. A very severe hemorrhage may lead to death in fifteen to thirty minutes.

Treatment. When a hemorrhage is not abundant, it suffices to take the animals to a moderately cool place, to protect them against every excitement and to give them complete rest; they should not be excited even by an examination. Hemorrhages occurring in young chickens may be stopped by bringing the animals to a warm place. If cough is present, it should be suppressed by narcotics, while the hemorrhage should be met by drugs which contract the vessels. Most commonly used are ergot (secale cornutum) (15.0-25.0 or 0.5-2.0; of the extract 5-10 or 0.2-1.0 gm. internally, the extract in half doses subcutaneously); ergotin (0.02-0.2 gm. hourly for dogs); extractum hydrastis (10-15 gm. for horses subcutaneously). Mellin and Plumier consider secale cornutum as not serviceable because it brings about contraction only in the larger circulation, so that it may increase the pulmonary hemorrhage; fluid extract of ergot is said to be an exception from this rule. In order to increase the coagulability of the blood, subcutaneous injections of Gelatina sterilisata pro injectione (Merck) may be given in doses of 40-60 gm. for large, and 2-10 gm. for small animals, also subcutaneous injections of a 1-2% aqueous solution of chloride of lime (at most 3-5 c. c. in one place, because of the danger of causing necrosis). In slow hemorrhage, both remedies may be administered by mouth (dogs 1 tablespoonful of a 4% solution of chloride of lime every two hours). Water containing vinegar, turpentine or chloride of iron are serviceable for inhalation. Cold packs of the thorax and, according to others, warm packs may be employed. If the site of the hemorrhage can be made out, the affected half of the thorax may be placed at rest by applying strips of adhesive plaster to it. One may also employ Moerkeberg's rubber plates (6-8 mm. thick), which may be moulded easily after a previous soaking in hot water.

In case of a unilateral dangerous hemorrhage of the lungs, physicians produce an artificial pneumothorax on the affected side in order to place the bleeding lung at rest.

Venesection appears indicated only in the presence of hyperemia of the lungs; excitants and digitalis must be used when great weakness is to be counteracted. Subsequent anemia must be properly treated. Animals which are predisposed to hemorrhage from the lungs must be protected prophylactically against excessive work, overheating, cold, etc.

3. Alveolar Emphysema of the Lungs. Emphysema pulmonum alveolare.

The term emphysema of the lung designates an abnormal dilatation of the alveoli with an enlargement of the lungs due to it; the pulmonary parenchyma may either remain normal (emphysema pulmonum alveolare acutum) or atrophy may occur of the interalveolar septa (emphysema pulmonum alveolare genuinum s. substantiale s. essentiale).

(a) Acute Alveolar Emphysema.

(Simple Bloating of the Lungs.)

Acute alveolar bloating of the lungs consists in simple dilatation of the air vesicles without any structural changes of the pulmonary parenchyma. It is always secondary in nature and may disappear completely as soon as the primary affection is relieved. It is therefore of minor clinical importance. According to the primary disease simple bloating of the lungs may be diffused over both lungs (volumen auctum pulmonum, Krehl) or it may be confined to portions of the lungs (emphysema pulmonum alveolare vicarians).

Etiology. Diffuse acute bloating of the lungs is a regular concomitant to diffuse microbronchitis. It is seen in the course of pleurodynia (see page 621); also in long-lasting convulsive cough, in long-continued strong inspiration and expiration as it is seen in certain diseases of the air passages or in a protracted agonal struggle. Marggraf saw several cases of the disease in cattle, kept during the winter in badly or not at all ventilated stables. Albrecht observed it as a sequel of puerperal paresis, probably because of the severe inspiratory and expiratory movements when the animals frequently attempt to get up at the beginning of the somniferous stage.

Circumscribed acute bloating of the lungs develops if the bronchi of a certain territory become narrowed or if certain parts of the lungs have become obstructed. In the former case the corresponding portions become emphysematous; in the latter case, neighboring areas.

Pathogenesis. Diffuse as well as circumscribed emphysema of the lungs may be produced by overdilatation of the alveolar

walls in case of long-continued forced inspiration and expiration as well as by convulsive coughing, because, on the one hand, the elasticity of the repeatedly overdilated alveolar walls decreases as in the case of any elastic body under similar conditions, and accordingly the expiratory contraction of the lungs becomes less complete, and on the other hand the air hunger inaugurates a new inspiratory movement even before the expiratory movement is completed. The result will be a gradually increasing emphysemic condition of the lungs. In a similar manner, the vicarious emphysema is produced in case of undisturbed access of air through the lung tissue bordering upon those parts of the lung which have already become inelastic. The disturbance of the free passage of air from the alveoli also plays an important rôle aside from forced breathing and the often repeated coughing in case of obstruction to the air currents within the bronchi, because during inspiration, which is aided by the active participation of the respiratory muscles, the normal amount of air will enter the alveoli, while during the following expiration a part of the inspired air will be retained in the alveoli on account of the relatively small power of the expiratory muscles and because of the increasing stenosis of the smallest bronchi following each expiratory contraction of the lungs.

Corresponding to the decreased elasticity, the ability of the lung tissue to distend, and particularly its ability to retract rapidly to its original volume, decreases. Furthermore, the blood current in the lesser circulation is impeded, because the resistance in the capillaries of the lungs (which, under such conditions, become longer and of a smaller caliber) increases, and the restricted movements of the thorax in pronounced emphysema does not aid the circulation in the same degree as during quiet respiration.

Symptoms. Aside from the symptoms of the primary disease there is in diffuse acute bloating of the lungs, an increased resonant percussion sound over the lower and posterior pulmonary margins and a displacement backwards of the posterior inferior pulmonary border, which may be extensive enough so that the posterior inferior pulmonary horder reaches to the costal arch (authors' own observation). The percussion souud is more rarely affected in circumscribed acute bloating of the lungs, i. e., when a larger lung territory has become affected. The respiratory sounds vary in both forms, according to the nature of the primary affection; if larger pulmonary territories have been obstructed one hears intensified vesicular breathing over the emphysematous portions.

The dyspnea of diffuse pulmonary emphysema occurs in variable degrees and may develop gradually or almost suddenly; in cattle it is accompanied by a moaning expiratory sound which can be heard from a distance. The animals stand with legs

spread apart; when they attempt to lie down from exhaustion, they are likely to suffocate and get up again immediately. As in all diseases associated with severe dyspnea, in acute pulmonary emphysema of cattle an interstitial emphysema is apt to supervene which increases the respiratory distress; crepitation and crackling sounds appear, and in some cases subcutaneous emphysema develops on the neck and in the region of the shoulders.

Course. If the primary disease is relieved rapidly, the acute pulmonary emphysema disappears promptly without leaving a trace; if the primary disease lasts some time, a chronic alveolar emphysema is apt to develop because the alveolar walls have undergone rarification. In cattle the course of the disease is often shortened by early killing.

Diagnosis. If the percussion sound is changed in the manner indicated above and if primary diseases as mentioned are present a diagnosis can be made safely, provided chronic alveolar pulmonary emphysema can be excluded. In cattle it may be necessary to exclude traumatic gastritis and pericarditis.

Treatment. This depends entirely upon the primary disease. Its removal must be sought for because its persistence entails danger of the development of lasting chronic dilatation of the alveoli. Under all circumstances, complete rest of the animal is indicated. In case of a disturbance of the air current in the smaller bronchi by catarrhal swelling of the mucosa or by the collection of mucus, repeated subcutaneous injections of atropin (for horses and cattle 0.3 to 0.05 gm.; for dogs 0.002 to 0.01 gm.) have a favorable effect by relaxing the bronchial muscles.

(b) Chronic Alveolar Emphysema. Emphysema pulmonum alveolare genuinum.

(*Heaves; Emphysema pulm. alv. substantiale.*)

Chronic alveolar emphysema consists in a permanent dilatation of the alveoli with a simultaneous partial obliteration of the interalveolar and interfundibular septa and blood vessels. It usually develops as a diffuse, true, genuine, substantial or essential emphysema, although more or less irregularly in the different portions of the lung and without showing other changes. Less frequently it develops as a vicarious or complementary chronic emphysema in different portions of the lungs in case of insufficient or arrested elasticity in the neighboring lung tissue.

Occurrence. Chronic emphysema of the lungs is found preferably in horses, more rarely in working oxen, quite fre-

quently in hunting dogs, usually in canines that are somewhat advanced in age. It is the most common cause of horses being broken-winded.

Etiology. In the majority of cases, chronic pulmonary emphysema develops primarily under the influence of violent inspirations and forced expirations if the animals are used for long periods for hard work or in rapid motion.

Secondarily the trouble often develops after diseases of the respiratory organs, if these lead to deep inspirations and forced expirations, lasting for weeks or months, or recurring frequently; also if they give rise to chronic cough. The most frequent etiological factor is continued and paroxysmal cough as it is frequent in chronic bronchial catarrh. Diffuse catarrh which involves the small bronchi and is associated with viscid secretion, is particularly dangerous in this respect because here the harmful effect of repeated attacks of coughing is added to that of the catarrhal narrowing of the bronchi. Accordingly chronic pulmonary emphysema follows frequently upon chronic bronchial catarrh which, however, may in its turn be a sequel of the emphysema. Ball saw chronic pulmonary emphysema in several cats with bronchial polyadenoma. Of the other respiratory diseases, chronic laryngeal stenosis is apt to produce true pulmonary emphysema, usually through paralysis of the recurrent nerve.

Emphysema of the genuine or substantial type may arise also from other causes aside from those already enumerated, and a permanent dilatation of pulmonary alveoli with atrophy of the pulmonary tissue may develop as a vicarious chronic emphysema of the lungs in various chronic diseases leading to the obstruction of portions of the lungs. This form is of clinical importance only in so far as it increases still further the respiratory difficulties caused by the primary disease.

Predisposition. The frequency of the disease increases with age. It is exceedingly rare in horses younger than five years, but common in older horses. The frequent occurrence of the disease in older animals finds its explanation in the fact that forced respiration continued over longer periods and due to hard work will produce a detrimental effect; work horses are moreover frequently exposed to affections of the respiratory organs and to disturbances of nutrition in consequence of insufficient feeding and of digestive anomalies.

The influence of insufficient nutrition is seen convincingly in senile atrophy of the lungs, where dilatation of the alveoli is due exclusively to atrophy and thinning out of the interalveolar septa. The power of resistance of the pulmonary tissue shows a good deal of individual variability because it would otherwise be unexplainable why some animals contract the disease after a comparatively short exposure to harmful influences and at a comparatively young age, while others

remain well under the same conditions or only develop a mild form of the affection late in life. A diminished resistance of the pulmonary tissue may be acquired, congenital or hereditary.

Pathogenesis. A considerable dilatation of the alveoli occurring again and again innumerable times, perhaps for years, will decrease the elasticity of the pulmonary tissue of itself to a certain degree. Much more important, however, is the circumstance that under the conditions indicated the alveolar pressure becomes increased during expiration or coughing and the interalveolar and interinfundibular septa with their capillaries, are compressed and distorted from both sides. Frequently recurring narrowing of the pulmonary capillaries impedes the free flow of the blood more or less and some capillaries will become impervious, while blood corpuscles are arrested in them. Since there is then no blood-current in some capillaries or at least only a current of blood plasma, the nutrition of the pulmonary parenchyma suffers, including that of *the compressed and distorted capillaries. For this reason and* in consequence of the ever increasing alveolar pressure the capillaries become completely obliterated, the elastic fibers yield to the increased pressure and the alveolar epithelia undergo fatty degeneration. Thus the interalveolar and interinfundibular septa gradually become thinner, the interstices between the yielding elastic fibers become larger, until finally the interalveolar and later also the interinfundibular septa disappear entirely so that neighboring alveoli become confluent to form a common cavity.

Once the development of pulmonary emphysema has set in, its further progress is favored by the exciting cause which is frequently still present, as well as by the circumstance that with each expiration a large number of bronchioles are compressed by the neighboring distended alveoli or infundibuli and in this way the escape of air from the alveoli communicating with such bronchioles is prevented (Ribbert). Another very condusive influence is exercised by the cough accompanying the bronchial catarrh which soon follows the emphysema and by a stenosis of the small bronchi.

It is believed in human medicine that pulmonary emphysema may be produced by a primary injury to the elastic lung tissue due to inflammatory toxic processes.

Ribbert, on the other hand, assumes in some of the cases a primary constriction of the bronchioles and of the alveolar passages which is said to be brought about by proliferation of connective tissue around the bronchioles and alveolar passages following certain types of inflammation of the bronchioles and pneumonia. Freund has shown that in certain cases a rigidity and expansion of the thorax following a peculiar degeneration of the cartilages of the ribs is the primary cause.

If this process goes on in the lungs it leads to a successive decrease of elasticity, hence the lungs will increase their size with difficulty during respiration and also decrease it with difficulty in expiration, so that a gradual increase in the volume of the lungs is produced. The obliteration of a portion of the pulmonary capillaries which bring about the exchange of gases, leads to a decrease of the respiratory surface, to an increase in pressure in the pulmonary artery and consequently to a hypertrophy of the walls of the right side of the heart. The decrease of elasticity and the diminution of the respiratory surface cause dyspnea which is particularly manifest in expiration. Deficient ventilation of the air passages, and changes in the lesser blood circulation predispose the animals to bronchial catarrh.

Anatomical Changes. The lungs appear larger, softer and less elastic (more like an air-cushion) and in consequence of the diminished blood-supply also paler than normal; their surfaces frequently show the impressions of the ribs (this is important from a forensic standpoint). The changes are most marked toward the inferior and posterior borders. The diaphragm appears raised posteriorly, the heart is covered by the lungs to an increased extent. The margins of the lungs are rounded off, individual air vesicles can be recognized with the naked eye and between them sometimes larger air spaces, up to the size of a hazelnut. If cut into, the lung tissue collapses more slowly than under normal conditions and without crepitation; the cut surface is pale red and discharges only a small amount of foamy blood; small droplets of pus, which appear on pressure, point to the existence of bronchial catarrh. The enlarged lungs are lighter in weight than those of healthy animals.

Stömmer has investigated the histologic changes and has ascertained that the diameter of the alveoli is increased from a normal of 0.15 mm. to 1.5 mm., while the diameter of the interalveolar septa is decreased from 8 μ to 1-2 μ. The vessels are more straight, their lumen is narrowed, partially obliterated. The number of elastic fibers is decreased, the alveolar epithelia are in a state of fatty degeneration. Rindfleisch found in the lungs of man that new vessels are formed partially in place of those that have become obliterated; this change is brought about by a new-formation from branches of the pulmonary artery which enter into anastomosis with branches from the bronchial artery. The involuntary muscle fibers of the bronchial wall are increased.

Hypertrophy of the right cardiac ventricle is found in advanced cases, in the most severe cases also dilatation with symptoms of general congestion.

In senile atrophy of the lungs (atrophia senilis pulm.) the picture is similar, because the alveoli have become enlarged secondarily, ex vacuo, in consequence of tissue atrophy. Such lungs are, however, never enlarged. They are, on the contrary, smaller and there is no hypertrophy of the right side of the heart since the obliteration of the lung tissue goes hand in hand with a corresponding decrease in the size and function of the other organs. This form of pulmonary emphysema is only seldom found in animals.

Symptoms. With the usual methods no changes can be demonstrated in the lungs while the disease is in its stage of development. On close inspection one may recognize a minor change in the respiratory movements; the expiration is somewhat prolonged and occurs with the auxiliary action of the abdominal muscles. In less well nourished animals one sees during inspiration a slight sinking in of the intercostal spaces and of the wall of the thorax in the cardiac region. The frequency of respiration is at first not changed. The animals are, however, more easily tired during work, and they breathe then more rapidly; they show in short the symptoms of being short of wind even at this early stage.

In a more advanced stage dyspnea becomes distinct. Expiratory dyspnea is particularly pronounced, during which the abdominal muscles are prominently brought into play and the period of expiration prolonged (according to Boedecker the ratio between inspiration and expiration may be as 5:8-9); furthermore, abdominal pumping can be observed. Respiration is frequently accomplished in two stages characterized by jerky action (subsultus); inspiration is immediately followed by a short, rapid collapse of the thorax, and this in turn, after a short interval, by a violent contraction of the respiratory muscles, which may also produce an upward movement of the spinal column. In severe cases the collapse of the thorax may begin in the posterior region and progress anteriorly so that a regular surging is brought about and the expiration may again set in at the rear before it has been completed at the front. The labored expiration is always accompanied by the formation of a depression along the costal arches, the so-called "Dampfrinne." An inspiratory dyspnea is also present; the ribs are strongly moved upward and forward and this movement is accompanied by a more or less pronounced sinking in of the intercostal spaces, of the more elastic lower portion of the thoracic wall, of the anterior aperture of the thorax, and of the flanks. Not infrequently inspiration is accomplished in two stages (see Fig. 117). Respiration is usually somewhat accelerated.

A lasting dilatation of the thorax progresses in like degree with the gradual dilatation of the lungs. The more prominently curved ribs produce a barrel-shaped thorax, a picture which betrays the character of the disease on first sight.

The percussion sound over the lungs is deeper and markedly loud. The increase in intensity is particularly marked over the inferior margins of the lungs, where a higher but duller sound is heard under normal conditions. The area of cardiac dullness becomes smaller or may disappear entirely. In severe cases one can also show by weak percussion that the posterior boundary of the lungs has been displaced backward and downward. (In healthy horses which are not overly fat one finds

the posterior boundary of the lungs in the iliac line where it is crossed by the seventeenth rib, in the ischial line by the fifteenth rib; in the shoulder line by the eleventh rib; and it passes into the lower horizontal boundary in the seventh intercostal space; in emphysema of a high degree, however, the lungs reach to the eighteenth rib in the iliac line, to the seventeenth rib in the ischial line, and to the twelfth or fourteenth rib in the shoulder line. [See Fig 117].)

Weak vesicular breathing is heard often, together with râles, rhonchi or whistling which may become apparent only on deep inspiration. The exacerbations of the existing bronchial catarrh, which are not infrequent, produce strong and frequent

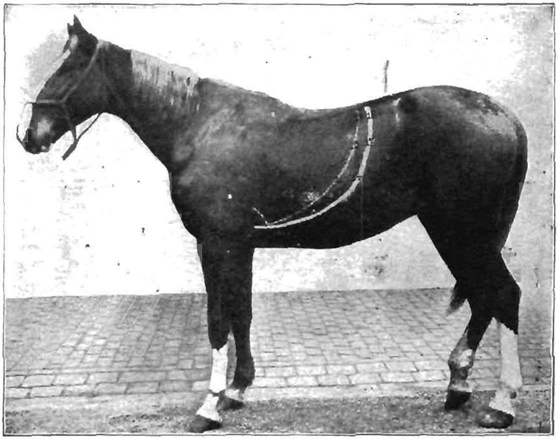

Fig. 117. Displacement of the posterior, lower boundary of the lung in chronic emphysema. The anterior finer line indicates the normal boundary which at C goes over into cardiac dullness: the heavier line indicates the boundary of the enlarged lung. The figures indicate the numbers of the ribs.

adventitious sounds and, in that case, the vesicular breathing is usually accentuated and rough over circumscribed areas. In this stage the symptoms of heaves are marked.

Cough is due to a more marked bronchial catarrh and usually occurs in a somewhat more advanced stage of the disease. It is weak in more pronounced cases of emphysema on account of diminished pulmonary elasticity, short, and as a rule, dull.

The apex beat is frequently somewhat accelerated, the heart sounds are weakened, the second pulmonary sound is commonly somewhat accentuated (felt in the median portion *of the left lower thorax in the third intercostal space). Transi*tory signs of cardiac weakness may come on, particularly after hard work.

The respiratory difficulties increase markedly during work and this reduces the work-value of the animals correspondingly with the advance of the disease, when the signs of shortness of breath become very marked.

The investigations of Richter and Schmidt have shown that shortness of breath produced by chronic alveolar pulmonary emphysema, particularly during exercise, brings about an elevation of temperature which will return to normal only after two hours. Half an hour after the exercise the temperature may still be 38.9° C.

Disturbances of nutrition and permanent circulatory disturbances appear after the disease has lasted a long time. Animals which were previously fairly well nourished or even fat gradually become emaciated; edema appears in the subcutaneous alveolar tissue, *in consequence of cardiac weakness.* Transitory cardiac weakness without edema may occur in an early stage, especially after severe work, but it disappears in a few days if the patients are allowed to rest.

Course. *Months, perhaps years, elapse until the disturb*ances in respiration and circulation point, even at rest, to material tissue changes. Holterbach saw severe shortness of breath appear forty days after a convulsive cough; it is not clear, however, whether this case was one of chronic emphysema of the lungs. The variable rapidity with which the disease develops is determined not only by the nature of the primary affection and by the degree in which it interferes with respiration, but depends also upon the manner in which the animals are cared for and used. Each physical exertion gives rise to excessively deep and accentuated respiratory movements, and also to transitory disturbance of the heart action, in consequence of which the disease process advances more rapidly. Under these conditions, and also because the animals catch cold or are kept in badly ventilated unhygienic stables, a bronchial catarrh is apt to develop which again tends to aggravate the condition. The disease naturally progresses more rapidly in horses which have come into the possession of poor owners, because of their diminished strength, where they are employed for hard work and are probably fed insufficiently. On the other hand, the emphysema usually develops very slowly in animals which are well cared for and are not exposed to overexertion, as is frequently the case in dogs, except those that are used for hunting.

Decided variations in the course of chronic pulmonary emphysema often are observed particularly in horses; they de-

pend upon the state of the bronchial catarrh which is usually present as a complication, and also upon the heart action. Every exacerbation of the bronchitis, no matter from what cause, or an accidental overexertion leading to transitory cardiac weakness gives rise promptly to rapidly increasing dyspnea, also to a displacement of the posterior lung border, which may go as far as the attachment of the diaphragm; acute pulmonary

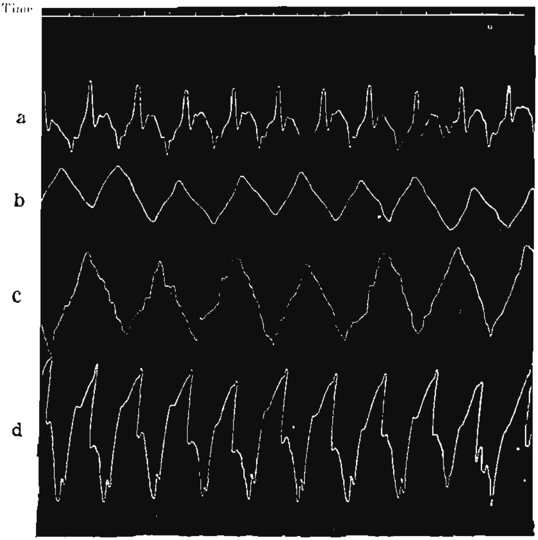

Fig. 118. **Pneumograph of a horse with pulmonary emphysema. a,** before injection of atropine (marked dyspnea with double expiration); **b,** 15 minutes after the injection of 0.05 gm. of atropine (dyspnea has disappeared); **c,** 1¼ hours after the injection; **d,** 17 hours after the injection (dyspnea again very marked).

emphysema, rarely an interstitial emphysema develops and causes the percussion note to become louder. If hygienic conditions are favorable, the previous condition is reëstablished gradually, sometimes in a few days, and the lung border may return to the former position. In this manner the periods of improvement may be explained which often are remarkable and may even simulate a cure, if the animals are allowed to rest in the barn, if they are turned out to pasture or are given green feed.

Usually true pulmonary emphysema is held to be an incur-

able disease because the dilatation of the lung cells necessarily maintains the respiratory disturbances and thus aggravates the disease.

Diagnosis. In a more advanced stage the disease can easily be diagnosticated, if the signs of enlargement of the lungs, of an increased air content, and a decrease of elasticity are present. *Early in the course of the disease it cannot be diagnosticated* by the aid of physical methods, because the changes in the percussion sound or the displacement of the lung borders are too slight. In an early stage of the disease the presence of pulmonary emphysema is suspected, if dyspnea (heaves) is noticeable during rest and particularly after motion, in cases in which careful physical examination of the thoracic organs fails to show anything abnormal, if a reliable history and examination justify the elimination of preceding acute bronchial or pulmonary disease or other affections with which dyspnea is associated.

Temporary relief from dyspnea is ofteo brought about by horse dealers by the internal administration of leaves, roots or seeds of plants containing atropine (Atropa belladonna, Datura stramonium, Hyoscyamus niger). Raitsits' experiments made in the Budapest clinic have shown that these plants do not merely reduce the frequency of respiration, but also abolish the double period and the forced character of the respirations, so that it may appear perfectly noroial, even in advanced cases. The effect is produced within fifteen minutes and lasts a whole day. After the effect has vanished the dyspnea becomes more intense than previously. For the first few hours the pupils are dilated and fixed, in some cases the vision is disturbed, the frequency of the pulse is increased, the mucous membrane of the mouth is abnormally dry, but attempts are made to hide this by applications of fat or oil. Subcutaneous injectioas of atropine (0.03-0.05 gm.) have the same effect, but this becomes manifest in a few minutes and persists only for 1-3 hours (see Fig. 118). The relief of the dyspnea is probably due to the relaxation of the bronchial muscles, through paralysis of the vagus, and also to the cessation of bronchial secretions, because of which the air can pass more freely through the bronchi. The use of atropin by quacks not infrequently leads to more or less severe or even fatal poisoning and distention of the stomach and intestines with gases, with or without rupture. In case the above-mentioned dose of atropin (in Weber's case 0.1 gm.) is exceeded and administered subcutaneously, bad results may follow.

Aside from its rapid development acute bloating of the lung is distinguished by the fact that it either comes on as a concomitant affection in acute disease of the lungs, or during hard work or after a long railroad transport, and that it disappears again after amelioration of the primary disease or during rest. It must, however, not be forgotten that the factors mentioned may also suddenly increase the symptoms of chronic emphysema or that their discontinuation may much improve the clinical picture of emphysema. Interstitial emphysema is characterized by a rapidly increasing dyspnea, without the possibility of demonstrating a displacement of the boundaries of the lungs, and frequently with the development of subcutaneous emphysema. Pneumothorax is characterized by a sudden onset, by a metallic percussion sound and by similar breathing sonnds.

A secondary bilateral pulmonary emphysema in focal affections of both lungs (tuberculosis, carcinomatosis; in Froehner's

case, sarcomatosis) can only be distinguished from the genuine emphysema in cases where the larger foci can be diagnosed clinically and permit conclusions to be drawn as to the secondary nature of the affection.

Prognosis. It is impossible to prevent the progressive development of the disease; the prognosis therefore is unfavorable as to complete recovery. Since, however, the development goes on very slowly, the animals may occasionally be serviceable for years for walking and for pulling moderate loads. The degree of the diminution of the ability to work must be ascertained by actual tests.

Every complication which appears during the course of the disease influences the prognosis unfavorably. This is particularly true of bronchial catarrh which is seen so frequently as a complication; its complete cure will be much retarded in consequence of the insufficient exchange of gases. Cardiac weakness is of similar unfavorable prognostic significance.

Treatment. Good nutrition and appropriate work may be relied upon in the absence of any specific curative treatment to prolong the period of usefulness of the animals. Complications which may arise, especially bronchial catarrh, must be properly treated (see page 590).

The systematic administration of arsenic (0.1-0.5 gm. pro die) is generally indicated in affections of this kind which are characterized by an asthmatic condition. The beneficial effect of arsenic depends on its improvement of the nutrition. The authors have found intratracheal injections of strychnine useless. Equally doubtful is the value of vergotinin (in teaspoon doses), a mixture of veratrine, 3 gm., strychn. sulph. 2 gm., ergotine cryst. 0.1 gm. and glycerine puriss. 150.0 gm., of which horses are given a coffeespoonful daily, for four weeks. The dose is mixed with a small quantity of oats, and covered with bran and oats. The favorable action which has been reported is probably due to the improved hygienic conditions which are a part of the treatment (rest or at most moderate work, diminished quantity of hay).

The same can be said of strychotin, which has a similar composition (0.066% ergotin, 1.29% strychnin and 1.93% veratrin in 96.71% solvent), with which Sustmann and Kuntschik had good results; also of strychnin, veratrin, and ergotin (Bengen). Diffiné, Bernhardt and Kleine report rapid improvement and even cures from the use of fibrolysin (thiosinamin and natrio salicylico) which they injected subcutaneously in horses 2 to 4 times in doses of 11.5 cc. at intervals of 2 to 3 weeks and following which they rested the animals for 2 days. Dornis, on the other hand, reports unfavorable results. (The fibrolysin

probably had a favorable effect upon the bronchial catarrh.) Wester had satisfactory results in horses following the administration of daily doses of 20 gm. calcium chloride. Uhland saw only temporary improvement following subcutaneous injection of atropin (5-10 cc. of a 0.1% solution daily. See page 637). Forssell and other Swedish veterinarians achieved decided improvement, sometimes even in apparently incurable cases, by the injection of 2 to 4 liters of sterile air into the pleural cavity, that is, with a kind of artificial pneumothorax (only in one particularly severe case, Forssell saw no improvement). Caution is indicated in the use of eserin and barium chloride in emphysematous horses suffering from other diseases (colic), since in consequence of the contraction of the bronchial muscles these drugs may greatly increase the dyspnea and even lead to suffocation, as is shown by the experiments of Raitsits.

4. Interstitial Emphysema of the Lungs. Emphysema pulmonum interstitiale.

Interstitial pulmonary emphysema consists in the accumulation of atmospheric air in the intra- and interlobular connective tissue of the lungs resulting from breaks in the continuity of the alveolar walls.

Etiology. Every considerable increase of the air pressure in the lungs, particularly if occurring suddenly, may directly lead to rupture of the alveolar walls. The advent of the condition is therefore observed frequently after *convulsive and forced attacks of cough*, in the course of acute bronchial catarrh, of bronchial croup, lungworm disease, also after the *entrance of foreign bodies into the air passages*, either accidentally or by aspiration. Pointed foreign bodies and lungworms may cause air to enter the lung tissue not only through intermediation of convulsive cough, but also by injuring the bronchial or alveolar walls; overexertion of the abdominal walls in pulling a heavy load, in delivery, in vomiting or in efforts of animals which have met with an accident. Rupture of the alveoli may also be brought about by continuous bellowing, by forced expiration, in rapid running, in excitement, in railroad transportation, in falling or kicking (Arendt saw a case of this kind in a horse), after traumatic insults to the chest, and finally *very deep and labored breathing in any disease*. Under these conditions, even the healthy lung tissue may tear but this takes place more rapidly, if the resisting power of the lung tissue is lowered pathologically, as in various lung affections, including chronic alveolar emphysema. Cattle are particularly inclined to interstitial pulmonary emphysema, probably because of the peculiar structure and slight distensibility of their lungs.

Michels, Detmers and, more recently, Royer, have reported on *enzootic in-*

terstitial pulmonary emphysema in cattle, in certain parts of Holland and Belgium (Maastal). This condition, called asthma, subacute pulmonary emphysema, pneumatosis bovum, probably had its cause in the catarrhs due to cold, which are frequent in these regions, also in the fog which is very dense during certain seasons and has a peculiar smell; it is possible that the fog and also irritating substances suspended in it, may give rise to deeper respiration and cough. The appearance of râles in the course of the disease, and the febrile rise of the temperature suggest an inflammatory origin. Joest suggests that the emphysema may be produced by lungworm disease. Knese observed an enzootic of the affection, associated with bronchopneumonia, in a herd of cattle pasturing on new grass; symptoms of cold were absent, and no further cases developed after the animals had been moved to older pastures.

An interstitial pulmonary emphysema is also the most prominent lesion in "jagziekte" of horses (crotalaria poisoning or crotalariosis equorum) in South Africa. The affection develops in 16 to 80 (usually in 50) days following the feeding of Crotalaria dura (daily at least 746 gm.). The development begins with a desquamation and necrosis of the alveolar epithelium, of toxic origin, followed by increased respiratory movements and minute ruptures in the alveolar walls. In the further course of the disease the irritation of the air penetrating through these ruptures leads to the formation of granulation tissue and new bronchioles.

Pathogenesis. If there are tears in the alveolar and bronchial walls, the tendency of the lung to collapse continuously forces air into the interstitial tissue; the entrance of air is intensified during inspiration by the suction, and during expiration and on coughing by the increased intrapulmonary pressure. The air which has entered the interstitial tissue is distributed in the form of very fine bubbles through the entire lung, the smaller air bubbles coalescing and forming larger ones. The air is moved toward the hilus, passes into the mediastinum and into the subcutaneous cellular tissue at the entrance of the chest from where it may spread over the entire body.

The air which has entered the interstitial connective tissue, causes the adjoining lung tissue to collapse, and thereby diminishes the respiratory surface; in its turn this leads to dyspnea which is the more severe, the more rapidly it develops; at the same time the elasticity of the collapsed lung tissue is diminished. The labored respiratory movements due to these circumstances may call forth variable degrees of acute distension of the lung portions which are not collapsed.

Anatomical Changes. Air bubbles varying in size from a nut to a fist, exceptionally also as large as a child's head, are found beneath the pleura and in the pulmonary parenchyma; the smaller bubbles are often present in large numbers and densely crowded. Otherwise the pulmonary tissue may be healthy or it may show the changes of a primary basic disease. The connective tissue of the mediastinum, of the chest aperture, of the neck and the subperitoneal tissue, may occasionally likewise contain air bubbles.

"Jagziekte" of horses can be distinguished from the above by the particularly large volume of the lung as a result of a serous subpleural imbibition of the greenish-yellow lung tissue. The surface of the lung is uneven and nodular, due to the presence of air bubbles, 3 to 5 cm. in diameter, especially in the anterior portion. Smaller air bubbles may also be found in the peribronchial and mediastinal lymph glands. Atelectatic foci of varying size with desquamated epithelium, fibroplastic tissue and newly formed bronchioles will also be found.

Symptoms. The air which has entered into the interstitial connective tissue soon compresses the neighboring alveoli; hence the respiratory surface becomes correspondingly diminished. *In some cases the disease therefore sets in suddenly* with dyspnœa which progresses so rapidly that the animals sometimes are cyanotic and threatened by suffocation after a few hours. The percussion sound either remains normal or

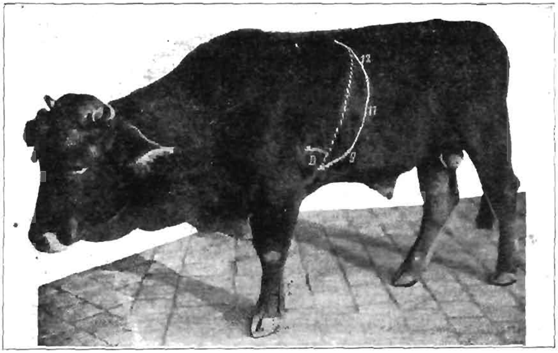

Fig. 119. Displacement of the posterior boundary of the lung in interstitial emphysema as a result of pulmonary tuberculosis. The dotted line indicates the normal; the continuous line the displaced boundary of the lung; *d*, dullness as a result of tubercular broncho-pneumonia. The figures indicate the numbers of the ribs.

is accompanied by tympanitic accessory sounds. In the presence of large subpleural air bubbles it may be purely tympanitic. The respiratory sounds, both in expiration and in inspiration, are occasionally accompanied by crepitant and cracking noises. In some cases, particularly in cattle, subcutaneous emphysema is developed subsequently and moderate pressure on the tense, *but otherwise healthy, skin elicits crepitation*; the percussion sound is in these places tympanitic. The subcutaneous em-

physema develops not infrequently, especially in cattle; it forms at the upper opening of the thorax, on the neck, in the region of the shoulders and may occasionally increase more and more, so that the whole body becomes swollen and disfigured. Subperitoneal emphysema can be felt with the hand introduced into the rectum. Acute distension of the lung tissue is a frequent complication and is recognized by the displacement backward of the posterior lung border (Fig. 119).

The condition is sometimes rapidly aggravated, so that the animals suffocate within one to two days, while in other cases even an extensive air infiltration disappears and recovery finally takes place after a considerable period of time.

Enzootic, interstitial pulmonary emphysema of cattle, which seems most frequently to affect cows toward the end of gestation, usually commences with fever up to 41.5°C. The pulse is rapid and weak; digestive disturbances appear in the form of fecal retention with or without meteorism. At the same time or soon afterward dyspnea commences and increases, very rapidly in severe cases, to attacks of suffocation, being associated with catarrhal sounds in the chest. Later, cutaneous emphysema usually makes its appearance. The disease lasts a few days, sometimes it disappears in a few hours, in other cases it terminates rapidly in death or often the animals are slaughtered.

"Jagziekte" of horses usually begins with a suddenly accelerated respiration up to 100 to 120 per minute and a less noticeable dyspnea. There will also be observed coughing and always intermittent, remittent or continuous fever (following feeding with Crotalaria, fever was also observed in the absence of pulmonary symptoms), in a later stage bronchial breathing as well as rales, and depressed appetite and depression. It runs its course in from 6 to 29 days. Recovery was not observed.

Diagnosis. The disease may be suspected if dyspnea has been preceded by the factors enumerated above; it can, however, only be diagnosticated beyond doubt when subcutaneous emphysema develops after the respiratory disturbances, and when other causes, such as injury to the upper air passages or to the esophagus, have been excluded, also in the presence of crepitation or crackling, possibly metallic sounds in both periods of respiration as well as synchronously with the heart beat; the cardiac dullness is sometimes obliterated. At its onset the disease may be confounded with hyperemia or edema of the lungs.

Acute edema of the glottis is differentiated by dyspnea associated with an inspiratory, whistling stenosis sound.

Treatment. To prevent if possible the further escape of air, complete rest must be enforced and cough, if present, must be alleviated with narcotics. Subcutaneous emphysema does not call for any special treatment, because the escaped air is

gradually absorbed spontaneously, provided that the tears have been closed; slight massage may hasten the absorption. Puncture must be avoided on account of the danger of infection. Hasenkamp, however, produced rapid recovery in a sheep which had become emphysematous all over the body by making a number of small incisions into the previously cleansed skin.

5. Croupous Pneumonia; Pneumonia crouposa.

(Pneumonia fibrinosa; Pneumonia lobaris.)

Croupous pneumonia is an acute febrile disease with a typical course, associated with the occlusion of the alveoli of larger continuous parts of the lungs, with an exudate consisting of fibrin and blood corpuscles. It occurs under the influence of various infectious agencies.

Croupous pneumonia is therefore not a uniform disease and cannot be differentiated sharply from catarrhal pneumonia, either etiologically or anatomically. The reason is that causes and microorganisms which usually lead to croupous pneumonia, may also give rise to a bronchopneumonia. On the other hand, bronchopneumonia sometimes is associated with copious secretion of fibrin and involves large lung areas in a short space of time.

Etiology. Croupous pneumonia is, in the last degree and without exception, caused by infectious agents. In some cases, however, other causes may at the same time play an important rôle in so far as they favor the development of the infectious agents in the lung tissue. Under some circumstances the importance of such secondary causes may even exceed that of the infectious agents, as without them the microorganisms would not be able to exert their inflammatory influences, and thus the impression may be given that the secondary cause is the sole cause of the pneumonia in such cases.

Croupous pneumonia of this type occurs as a localization of certain specific infectious diseases (swine plague, horse influenza, contagious pleuropneumonia of cattle) and it appears more or less widespread according to the pathogenic characters of the particular causative microorganisms. Microorganisms which are morphologically and culturally identical with such disease producers are found not uncommonly as saprophytes in the air passages or in the environments of domestic animals without interfering with their health. If, however, the power of resistance of the organism as a whole or of the pulmonary tissue alone has been diminished by definite external influences or by other diseases, such microorganisms are enabled to penetrate into the organs of the body, to multiply in the lungs and to produce an inflammatory process in them. Their virulency may so increase that they are able to invade healthy animals without the auxiliary effect of external influences. It cannot be denied, on the other hand, that microorganisms living as saprophytes

outside or inside of the animal organism may become pathogenic under special circumstances, and may be able to attack an animal the power of resistance of which has not been previously lessened. Such bacteria which have become pathogenic may lose their virulency in the diseased animal, or they may, on the contrary, retain it and spread the disease.

As secondary causes, there should first be mentioned external influences such as cold (also following inunctions in skin diseases), inhalation of irritating fumes (which may have a direct inflammatory action), overexertions of all kinds (as in long-continued driving, railroad and boat transportation, fruitless attempts at rising by weak animals, and also following tying up of the head and casting for an operation), contusions of the thorax, and permanent stabling in insanitary barns (stable pneumonia). Some of these influences have a directly harmful effect upon the resistance of the lung tissue, but as a rule they decrease the resistance of the organism as a whole by unfavorably influencing the phagocytic and bacteriolytic powers of the animal. Other pathologic conditions and disturbances of nutrition causing a decrease in the resistance of the body are also secondary causes.

The animal experiments by Duerck, Aufrecht and Jezierski have thrown some light upon the significance of cold in the origin of pneumonia and diseases of organs in general. Following a partial or general chilling of the surface of the body, small thrombi are found in the blood vessels of the lungs, due to a coagulation of the fibrin, and the permeability of the capillaries is increased also in other organs (pharynx, larynx, mucosa of the trachea), probably as a further result of the colloidal changes in the cells in the region of the chilling, as described by Schade. Similar changes will also be found in the liver and kidneys following chilling of the abdominal wall. Accordingly, small hemorrhagic foci will be found, which may be of larger size in the lung tissue. Due to disturbed circulation of the blood and the simultaneously impaired capillaries, the resistance of the lung tissue with respect to the microorganisms is greatly decreased, while the escaped blood forms a favorable medium for their multiplication (Duerck always found bacteria in the inflamed lung tissue resulting from artificial chilling of the body). The chilling will, furthermore, have a harmful influence upon the protective apparatus of the organism and in this manner indirectly upon the lung tissue. Transmission of the influence of the cold to distant parts through the sympathetic nervous system, setting up disturbances in the distribution of the blood in the internal organs, and secretory anomalies in the endocrine glands, may play a certain rôle.

According to the above, and judging from results obtained upon bacteriological examination, the occurrence of an etiologically specific, genuine croupous pneumonia, sui generis, has

not been proved. The repeatedly reported differences between this and other forms of pneumonia are not infrequently determined solely by a changing virulence of the causative organisms and the varying resistance of the animal. This is also supported by the fact that pneumonia which at first apparently occurs as a genuine form later takes on an epizootic character and exhibits all the symptoms of a specific pneumonia. Croupous pneumonia in man also occurs partly sporadically under the influence of secondary causes, and partly endemically, in spite of the fact that in both cases it is caused by the same *microorganism.*

In order to avoid misunderstanding, one should avoid the etiological segregation of a genuine pneumonia as a special form of disease, and reserve the term "genuine," or, better, "primary croupous pneumonia," as is done in human medicine, solely for the designation of a pneumonia occurring independently of previous diseases, in contrast to "secondary pneumonia," which tends to follow diseases which weaken the organisms. If it is possible to prove the specific origin of the otherwise primary or secondary inflammatory process, then the general designation "specific croupous pneumonia" would be applicable to the forms belonging in this category (influenza of horses, contagious pleuropneumonia of cattle, etc.), while the term "common croupous pneumonia" should be used for the other forms of pneumonia.

Particularly in the horse, croupous pneumonia frequently occurs primarily as an influenzal pneumonia (see volume 1). Several authors (Roell, Froehner and Zwick, Siedamgrotzky) believe in the occurrence of a genuine croupous pneumonia aside from influenza, while according to still other authors (Lustig, Schuetz, Dieckerhoff, Malkmus, Cadéac) all forms of croupous pneumonia of the horse would have to be classified as influenza. *According to the foregoing general remarks* as to the etiological character of these ailments, an etiologically segregated genuine pneumonia with a typical clinical course can not be separated from influenza, even when an introduction of pneumonia from other sources can not be shown and also does not show a contagious character. In fact, the so-called genuine pneumonia exhibits just such characteristics in its occurrence as those which alone permit the drawing of conclusions as to its influenzal nature. Thus in almost *all large epizootics of influenza it can be observed that while* some of the horses manifest unmistakable symptoms of influenza, others in the same barn and under identical conditions show exclusively a typical croupous pneumonia. It has also repeatedly been observed that a horse suffering from so-called genuine pneumonia infected its neighbor and even all horses in the barn, which latter animals then showed typical symptoms of influenza. Furthermore, sporadic cases of typical influenza

are not infrequently observed. In this respect the observation made at the Budapest Clinic seems significant. After influenza had been eradicated from the stables in the city, no more cases of typical croupous pneumonia were brought to the clinic for a period of two years. Accordingly, no hard and fast line can be drawn between alleged genuine croupous pneumonia and influenza.

Croupous pneumonia not infrequently follows external influences which act as secondary causes and which, as a rule, also lead to the development of influenzal pneumonia by lowering the resistance of the animal or of the lung tissue for the causative organisms of influenza which are accidentally introduced. On the other hand, the development of pneumonia under the influence of other specific or common infectious organisms can not be denied, but in such cases the pneumonia usually shows an atypical and unfavorable course. Corresponding to the particular uses to which horses are put and their sensitiveness to harmful effects, external influences play a rôle as causes of croupous pneumonia which may be more important than the causative organism itself.

Croupous pneumonia occurs secondarily to catarrhal influenza and epizootic laryngo-tracheal catarrh, in the course of strangles and pharyngitis, hemorrhagic septicemia, purpura hemorrhagica, and following severe gastro-intestinal catarrh or enteritis, or long-continued colic. It is also observed in long-continued weakening diseases and severe exhaustion resulting from permanent insufficient nutrition. These forms of pneumonia frequently show an atypical course.

The occurrence of croupous pneumonia independently of pleuropneumonia in **cattle** is established beyond doubt; it develops usually after the invasion of bacillus bovisepticus (see Vol. I). It is also commonly observed as a foreign body pneumonia after the entrance of foreign bodies from the air passages or from the forestomachs, although it does not show a typical course in these cases and usually takes the course of a catarrhal pneumonia.

The observation of Jensen, Buch and Krüger have shown beyond a doubt that hemorrhagic septicemia of cattle also occurs sporadically. Schütz found "ovoid" bacteria in the affected lungs in croupous pneumonia of cattle. Krüger demonstrated the presence of bipolar bacteria by inoculation experiments. Coulon and Olivier saw croupous pneumonia in cattle in wet valleys; Cagny in steers which were kept in the open during cold weather. These affections, however, could not be transmitted by application of the expressed lung juice to the skin denuded of its epithelium, or by subcutaneous application with a vaccination lancet. Nevertheless, the negative result does not exclude the possibility of pneumonia being caused by the bipolar bacterium, because subcutaneous inoculation is not always successful even when lymph of animals is used which undoubtedly suffer from the pectoral form of hemorrhagic septicemia.

Hogs suffer from the pneumonic form of swine-plague which often assumes a croupous character (see Vol. I). The cases of pneumonia observed by Bayer in Hungary probably belong to this type. Hemorrhagic septicemia which sometimes spreads from cattle to hogs may likewise form the basis of a croupous pneumonia if it does not end in death rapidly. Since bipolar bacilli with the morphologic characteristics of bacillus suisepticus occur in the buccal cavity, pharynx and nose of the hog, they may get into the bronchi with the feed, in greedy feeding or with accidentally inhaled foreign bodies, and may there cause a foreign body croupous pneumonia. The observations of Passerini and Wyssmann prove that anthrax in hogs may lead to a croupous pneumonia; the latter may form the exclusive localization of the anthrax infection.

The epizootic pneumonia which is rarely croupous in **sheep** is caused by bacillus ovisepticus (see Vol. I); the infectious pneumonia of anatolic goats (see Vol. I) is likewise due to bipolar bacteria; the cause of infectious pleuropneumonia of goats is unknown (see Vol. I).

According to older statements (Röll, Trasbot, Boissière, Renault) **dogs** were said to be frequently subjects of croupous pneumonia. These older statements, however, do not deserve much credence because the cases referred to were evidently distemper bronchopneumonias, which not infrequently involve whole pulmonary lobes. One may claim this with confidence, since the recent reports concerning croupous pneumonia of carnivora are very meager and the few cases published refer only to clinical observations. Distemper bronchopneumonia leads to only a partial formation of fibrinous exudate.

Recently Lamar & Meltzer succeeded in producing a typical croupous pneumonia in dogs by injecting pure cultures of Diplococcus pneumoniae deep into the bronchi. In the fatal cases, and also clinically, the disease was quite similar to pneumonia in man; but the non-fatal cases took an atypical course in so far as a typical fall of the fever temperature occurred as early as 36 to 48 hours, although the pneumonic process made further progress.

According to Friedberger & Fröhner **cats** suffer not infrequently from croupous pneumonia. Whether this disease is identical with the cat epizootic observed recently by Gärtner in Greifswald, cannot be decided definitely. The latter disease is, according to Gärtner, caused by the bacillus pneumoniae felis, a microorganism belonging to the group of bipolar bacteria; the affection consists of an extensive necrotizing pneumonia, principally of the posterior lobes; it is sometimes accompanied by a hemorrhagic, fibrinous pleurisy. This bacillus is probably identical with one described by Marx (bac. pneumoniae tigris) and found in a tiger dead from hemorrhagic pneumonia (Gärtner). Typical croupous pneumonia in the anatomical sense is also found in carnivora ill with glanders (Kitt).

Rabbits suffer from croupous pneumonia after an attack of

contagious rhinitis (see page 536), or this affection represents the localization of such an invasion. Südmersen described a bacillus of the colon group as the cause of an enzootic pleuropneumonia of rabbits; Selter, one of the bacillus bipolaris septicus group (compare catarrhal pneumonia).

Croupous pneumonia in **fowls** is seen in slow cases of fowl cholera. Jowett saw an epizootic pneumopericarditis in turkeys, which was caused by the bacillus bipolaris septicus.

For details as to these affections due to bipolar bacilli the reader is referred to the first volume, dealing with infectious diseases.

In man, genuine pneumonia is in the majority of cases caused by the Diplococcus pneumoniæ (pneumococcus), which, however, is not infrequently also found in secondary pneumonia either alone or together with other bacteria. A typical form of the disease can also be produced in the lower apes by the intratracheal injection of cultures of pneumococcus and may then become contagious. Spontaneous or epizootic diplococcus pneumonia is also observed in monkeys kept in captivity (Blake, Francis and Russell). Now and then genuine pneumonia also occurs under the influence of streptococci and Bacillus pneumoniæ and it may also be produced artificially in monkeys by an intratracheal injection of cultures of Streptococcus hemolyticus. Many observers consider Diplococcus pneumoniæ and Streptococcus pneumoniæ as identical or as variants of the same bacterial species. The organisms of primary infectious diseases (diphtheria, influenza, typhoid fever) have also been found in secondary pneumonia.

Susceptibility. On account of the great prevalence of influenza, horses are most commonly affected with croupous pneumonia, as far as domestic animals are concerned. It is not merely relatively but absolutely frequent among horses and follows in frequency the colicky diseases. Younger, well nourished horses are most commonly affected; poorly nourished and overworked horses are less susceptible; young foals still less. Other domestic animals are affected more rarely or not at all by this form of pneumonia.

Pathogenesis. The bronchi are frequent ports of entry for the infectious virus; microorganisms invade the alveoli of the lungs after having entered the bronchi accidentally, by being aspirated from the pharynx during inspiration, with particles of mucus, of drinking water or of food. On the other hand, a hematogenic infection is perhaps not an infrequent occurrence, the infectious material being absorbed into the circulation from intestines or pharynx; in favor of this mode of infection are the digestive disturbances, which occasionally precede the symptoms of pneumonia, and also those animal experiments

in which it was possible to produce a croupous pneumonia by the subcutaneous injection of appropriate bacteria. In many cases a lymphogenic origin cannot be denied, the microorganisms being transported to the lymphatic apparatus of the pharynx, and from there to the bronchial lymph nodes and to the lymph nodes of the hilus of the lungs. In the lung tissue the infectious germs are disseminated, by their rapid multiplication, along the bronchial mucous membrane and to the alveoli, also by aspiration into the smaller bronchi and alveoli, but most largely through the lymphatic circulation after having been absorbed from the bronchial mucous membrane. Microorganisms which have been carried into the lungs accidentally, by the blood or the lymphatic circulation, partly enter the alveoli and smaller bronchi, also partly invade the lymph spaces of the interstitial tissue and are then distributed similarly as in cases in which the infection proceeds from the bronchi. The usual multiplication of the bacteria in the ventral and cranial lung portions in animals, and in the caudal portions in man, appears to prove that the dissemination takes place mainly through gravity along the bronchial lumen; but this difference and also the most frequent localization may quite well depend upon the mechanism of respiration and upon the flow of the lymph.

The pneumonic process commences with marked engorgement of the affected lung areas, after which a fluid exudate, highly albuminous and mixed with many red blood cells, is excreted into the alveoli and the adjoining small bronchi which thereby are deprived of air. At the same time the alveolar epithelia swell up and are in part imprisoned in the exudate which coagulates, forming a fibrin-net of variable density (red hepatization). As the hyperemia gradually diminishes during the succeeding days, increasing numbers of polynuclear leucocytes migrate (gray hepatization) which, because of their high enzyme content, cause the disintegration of the fibrin together with the other protein substances and the albumin of the cellular constituents of the exudate. In this manner they become responsible for softening and fatty degeneration of the exudate (yellow hepatization), and finally for its liquefaction (resolution). On the other hand, from the first, the leucocytes ingest the red blood cells which break down immediately on leaving the blood vessels, also other formed elements as well as a part of the bacteria, and carry them into the circulation where the ingested cells are dissolved in greater part. The liquefied exudate which contains a great many split products of various protein substances, is absorbed mostly into the circulation, and only a small portion of it is removed with the expectoration.

From the very beginning the causative organisms are partly dissolved by the normal lysins of the leucocytes and body fluids, and the endotoxins thus liberated produce the inflamma-

tory changes in the lung tissue. This struggle of the animal body begins simultaneously with the multiplication of the causative organisms in the lung tissne, and continues until finally all organisms have been destroyed by the specific lysins (antibodies) which have been liberated under the influence of the endotoxins either in the lung tissue itself or in the blood-forming organs, especially the bone marrow, and which are now carried to the diseased foci by the blood stream. In the destruction of the microorganisms, bacteriolytic products of autolysis in the exudate (soaps, according to Flexner and Lamar) probably play a rôle. According to the resistance of the microorganisms and the congenital or acquired lysin content of the animal on the one hand, and the reactivity of the blood-forming organs on the other hand, the dissolution of the bacteria takes place in the first few days, or only after several or many days, or they are not dissolved at all, as in cases where foreign bodies, necrotic tissue, etc., remove the microorganisms from the sphere of action of the specific lysins. These conditions also explain why many cases of pneumonia caused by microorganisms which were originally very virulent do not terminate in lysis, or only seldom do so, and why the same termination of pneumonia is frequently observed in the presence of foreign bodies or necrotic foci, as well as in emaciated weak animals. Although this solution proceeds in greater part in the blood stream, the liberated endotoxins enter the general circulation, together with the disintegrated products of the proteins, and give rise to fever which subsides by crisis or by lysis, accordingly as the disintegration is rapid or gradual (Wolff-Eisner). If a portion of the bacteria happens to escape destruction, they are dissolved after more lysins have been formed, and in this manner a renewed *temperature-rise is caused. A permanent fall of the* temperature is possible without the complete disintegration of all bacteria, if the absorption in the diseased focus is arrested.

The bacteria which enter the blood stream during the entire period of inflammation, especially during the period of resolution, or any bacteria which enter in consequence of a secondary infection, may in part escape bacteriolysis and are deposited in organs, leading to inflammatory processes either immediately or through the agency of favoring circumstances. In pleurisy, which is a frequent complication, the pleura is affected either by continuity, or the bacterial toxins and the bacteria themselves reach the pleural surface by way of the lymph stream. It is possible that the bacterial toxins and the products of disintegration of the exudate, which circulate in the blood, are deposited and may then, in certain organs, be sufficient to produce inflammation. Aside from such a bacteremia, a septicemia may supervene in certain cases with permanent increase of the bacteria and of the secondary associated microorganisms in the circulating blood.

The disintegration of the protein substances, and particularly the endotoxins of the broken-down microorganisms, produce an injury to the parenchymatous organs, primarily of the kidneys which eliminate these substances, and also of the heart muscle. The vasomotor apparatus is injured more or less, partly by the direct action of the products of disintegration, partly by the prolonged increase of function, but more particularly when resolution is impending, at which time large amounts of products of disintegration are taken up into the circulation. The blood circulation is influenced adversely to a noticeable degree, by the diminution of the lung surface which intermediates the gas-metabolism, but this does not occur until extensive lung portions are deprived of air, inasmuch as the stimulating influence of the respiratory movements upon the pulmonary circulation is decidedly diminished, and the circulation is slowed in the inflamed territory.

Anatomical Changes. The anatomical changes become manifest in a large continuous portion of one or of both lungs, usually in the lower and anterior parts. The inflammation is rarely limited to the hilus-portion or to the caudal lobe, and very rarely the upper parts of the lungs are involved exclusively. During the stage of congestion (stadium hyperaemiae) the affected lung tissue is brown-red, of diminished elasticity, not infrequently containing hemorrhagic foci of variable size. When the exudate coagulates (stadium hepatisationis) the diseased part of the lung is enlarged, firm like liver tissue, friable; in water it sinks to the bottom. The cut surface usually is finely granular, rather dry, at first reddish-brown (stadium hepatisationis rubrae); during the following days the color gradually turns grayish-red (stadium hepatisationis griseo-rubrae), then light-gray (stadium hepatisationis griseae), and the lung tissue becomes softer and less dry. With the gradual fatty degeneration of the exudate, a yellow color appears (stadium hepatisationis flavae). In accordance with the variable rapidity of the process, the cut surface may assume a mottled appearance like granite, patches with red hepatization being found with gray or yellow hepatized spots. The mottling of the cut surface becomes more evident if the interstitial tissue is subject to gelatinous or cellular infiltration. It is found particularly in croupous pneumonia of cattle and hogs; in cattle it may be mistaken for contagious pleuropneumonia (see Vol. I). After the exudate has become liquefied (stadium resolutionis), a reddish, rather thick fluid may be scraped from the cut surface, and is mixed with fine air bubbles and droplets of fat; at the same time the softness of the lung tissue is increasing. In the infiltrated area, microscopical examination shows the alveoli filled with a fine fibrin network, the spaces of which contain leucocytes and numerous red blood cells, especially at the beginning. During resolution, one sees detritus

and fat globules; the interstitial tissue shows small-cell infiltration. In many fatal cases abscesses or necrotic foci are seen in the diseased areas. The catarrhal bronchi of the inflamed lung portions usually contain mucopurulent material, the smaller bronchi sometimes are filled with fibrin-plugs or with fibrinous membranes.

The visceral pleura over the affected pulmonary portions is almost always lusterless, cloudy or rough, and occasionally infiltrated with small hemorrhages. Not uncommonly a fibrinous or serofibrinous pleurisy is found. The bronchial glands usually show acute swelling. The post-mortem examination also shows secondary changes, such as cloudy swelling and sometimes fatty degeneration of the parenchymatous organs, especially of the myocardium and the kidneys.

Symptoms. The period of incubation of the specific pneumonia is similar to that of influenza pneumonia, but under certain conditions it may be considerably shortened by secondary causes. Ordinary primary pneumonia develops in the first few days following exposure to known external influences. Thus, in a case observed by the author in a very spirited horse which had become quite hot following a drive of one hour, light fever with all the usual phenomena appeared one hour after unharnessing. In the course of another three hours the fever *had risen to 40.1° C. (104° F.), and seventeen hours after the* appearance of the fever, tympanitic percussion sounds, partly loud and partly dull, could be detected in the region reaching up to the level of the shoulder joint and back to the eighth rib. The pneumonia took its usual typical, favorable course. In secondary pneumonia no real period of incubation can be determined, since it may develop at any time, and sometimes also from day to day in the course of the basic disease.

Except in very rare cases the disease begins with the symptoms of a febrile condition with sudden onset. The appetite, *and in ruminants also rumination are diminished; the former,* however, is only rarely suppressed entirely. The patients stand listlessly and with drooping heads in front of the crib, smaller animals like to hide themselves and lie continually on the floor. Frequently one even observes at this stage cough and an accelerated respiration. The temperature rises to 40-41° C. and above within half a day and in some patients chills and muscular tremors occur. In older and debilitated animals the temperature does not show a considerable rise, but the other general symptoms reach an intense degree. If, for some reason or other, *the animal already had fever, the onset of pneumonia is indicated* by a sudden aggravation of the general condition and possibly by an additional rise in temperature.

After the fever has lasted from one-half to two, exceptionally also from 3 to 4 days, changes in the percussion and

respiratory sounds become manifest, which can now be watched in modifications characteristic for the disease.

Percussion gives at first a somewhat dull sound, which assumes a tympanitic timbre usually on the second or third day, rarely earlier, and soon becomes purely tympanitic or it may also become gradually less intense. The change in sound usually shows itself at first in the region behind the elbow and spreads from there backward and upward to an extent which varies from case to case. As the alveoli become more and more consolidated, the tympanitic sound gradually changes into a dull tympanitic and later on into a very weak, dull sound. The area of dullness varies as to size and boundary line. It usually extends backward from the elbow and reaches to the middle or even to the upper third of the thorax. Usually, although not always, the upper boundary line describes a curved line, with the convexity above, or descending backwards (see Fig. 120). In cattle, the changed percussion note may be shown

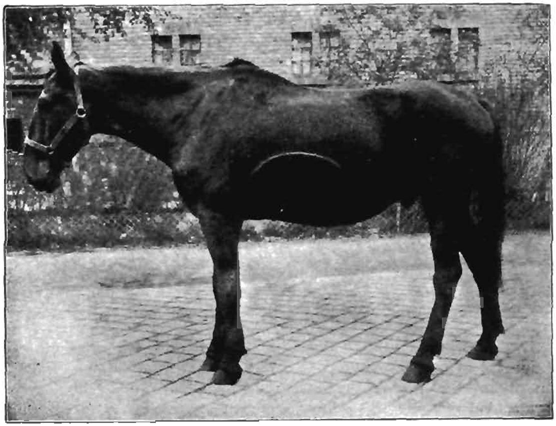

Fig. 120. Arched boundary of area of dullness in croupous pneumonia.

also in the prescapular field, after the fore-foot has been pulled back. It remains unchanged for three to five days, when the sound again assumes a tympanitic timbre, later on it becomes purely tympanitic and then gradually changes again into a normal, loud, non-tympanitic percussion sound. The low, dull sound only rarely changes to the normal without having first assumed a tympanitic timbre.

Deviations from the changes here described are observed. In affection of the deeper portions of the lungs the percussion sound may either remain normal or not change until later on *after the inflammation has reached the external strata.*

If the consolidation of the upper boundary of the affected area is confined to the deeper strata, one hears above the upper boundary of dullness a tympanitic sound which may *exist for several days. If the deeper layers are consolidated* in such a manner that they nowhere touch the wall of the thorax, but are separated from the wall by considerable portions of healthy lung tissue, one hears only a tympanitic sound

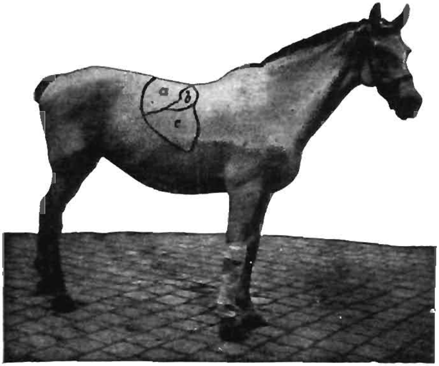

Fig. 121. Croupous pneumonia with an atypical location. *a*, area of dullness over the most posterior and upper portions of the lungs; *b*, area of a high, and *c*, area of a low tympanitic sound.

during the whole course of the disease, and this changes into a normal percussion sound during the stage of resolution. On the other hand, the changes in the percussion note, which have been mentioned, sometimes are limited to the posterior and upper borders of the lungs (Fig. 121).

During the initial stage of the disease auscultation reveals intensified and rough vesicular breathing on account of the swelling of the bronchial mucosa and of the accelerated respiration; even then high and crepitant râles may, however, be heard, especially during inspiration, indicating the presence of a serous, thick exudate in the alveoli and bronchioles. When a tympanitic or weak percussion sound is audible, bronchial respiratory sounds are usually heard, and frequently also metallic râles which usually disappear promptly; during resolu-

tion, crepitation and râles reappear, and gradually vesicular breathing becomes manifest. If the area of dullness becomes larger and the lumen of the smaller bronchi also becomes filled with an exudate there is usually an absence of all breathing sounds. Even in these cases, vesicular breathing can usually be heard at the borders of the dullness or higher up, where the hepatization frequently involves only the deep lung portions, and from these the bronchial respiration is transmitted by the air-containing tissue, in contrast to the lung portions which are situated more centrally and are wholly infiltrated; in these the smaller bronchi often are filled with exudate and therefore prevent the transmission of bronchial respiration to the surface of the chest. It may occur also that bronchial breathing can be made out only in certain places during expiration or during deep breathing. These modified respiratory sounds usually can be discovered only by repeated systematic physical examination. This fact, and perhaps the circumstance that, in certain enzooties, the smaller bronchi are involved more frequently, may account for Trautmann's assertion that bronchial breathing during hepatization, and râles during resolution of croupous pneumonia in horses are infrequent, while other observers claim the contrary; bronchial breathing could be heard in 75% of the authors' cases.

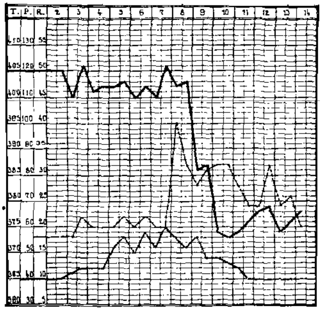

Fig. 122. Fever curve in croupous pneumonia of a horse. Crisis. Considerable increase in the pulse and acceleration in respiration occurred with the approach of the stage of resolution.

Of like significance as the bronchial breathing is the accentuated bronchophony which renders groaning, coughing, and the voice more audible and sonorous, also the stenosis sound which is produced artifically by compression of both arytenoid cartilages. The same is true for the tracheal percussion note on the thorax, if an assistant percusses the pleximeter which is held firmly at about the middle of the lower border of the neck, while the examiner auscultates the thorax. According to Schindelka, Wirth and Babor, the short and sharp tracheal sound is heard clearly over purely pneumonic areas, as though it originated immediately beneath the ear; in healthy animals it is attenuated and indistinct as if coming from a distance. In the authors' experience, this change in the tracheal sound in

pneumonia is not heard in cases and at places where the conditions are not favorable for bronchial breathing to be heard.

A rusty or saffron-colored nasal discharge is seen only in a minority of cases; it then comes on almost always before the beginning of hepatization and persists either only one or two days or until resolution occurs. A saffron-colored nasal discharge coming on later usually points to the development of new infiltrations. It is, however, a very valuable symptom, since it is frequently the only symptom in central pneumonia which may afford information concerning the cause of the fever and the nature of the disease.

The respiration shows a normal costo-adominal type; it is, however, more or less forced and accelerated, according to the extent of the consolidation; the acceleration is least marked in horses in uncomplicated cases. Nieder saw Cheyne-Stokes respiration in a horse persisting for two days. Cough is present from the start; it occurs, however, only at long intervals or after external stimuli, for instance, after percussion. In the stage of hepatization it appears painful, hence less violent, even very feeble, but it becomes markedly easy and moist with the advent of resolution.

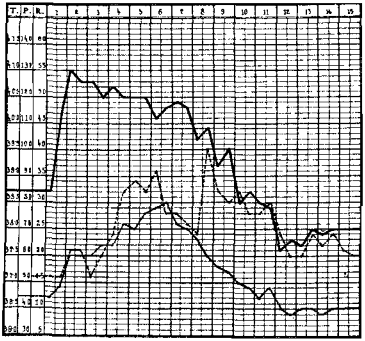

Fig. 123. Fever curve in croupous pneumonia of a horse. Lysis. Gradual increase of the frequency of the pulse and respiration with the approach of the stage of resolution.

The course of the fever is usually very characteristic for the disease in those cases which run their course without complications.

The temperature rises on the first day to 39.5°-41° C. and remains high with slight variations during the next days (febris continua—see Figs. 122 and 123). Toward the end of the stage of hepatization, i. e., toward the end of the first week, the temperature either falls rapidly within ½ to 1½ days (crisis —Fig. 122) or gradually with remissions in the morning and exacerbations in the evening, so that the animal is free from fever only after 2 to 5 days (lysis—Fig. 123). The fall in temperature is usually preceded by a change of the dull and low percussion sound into a tympanitic or loud sound.

The temperature, which has fallen to normal or which is falling rapidly, may, in exceptional cases, rise again on the following day; it remains, however, high only for two days and then again goes down (perturbatio critica). Exceptionally the temperature may fall below normal, occasionally down to 36° C.; this occurrence, however, is likewise of short duration. The authors have seen intermittent fever in horses, as a rule, in combination with other severe symptoms.

The heart's action does not go parallel with the fever; there is, on the contrary, a certain antagonism between the fever curve and that of the pulse beat. The latter is only moderately accelerated in the beginning, in spite of the high fever (in horses up to 50 per minute) and the pulse is tense and full. The contractions of the heart become, however, more frequent in the latter course of the disease, and at the same time less strong; the pulse becomes empty and small (Figs. 122 and 123). This cardiac disturbance becomes the more marked the larger the affected lung territory and the more intense the infection. In the less severe cases the heart's action becomes again normal as soon as the stage of resolution is completed; on the other hand, there may be arhythmia or allorhythmia of the heart; it is, however, usually of no significance even if it still exists during the stage of convalescence.

The general condition and with it the appetite of the animals varies a good deal from case to case. In milder cases the animals may ingest a normal amount of feed, though more slowly, during the whole course of the disease; others may refuse feed entirely during the course of the fever, and may show a considerable degree of listlessness and prostration. Sick horses and cattle usually remain standing during the whole course of the disease; ponies and the smaller domestic animals lie down a good deal; in the case of unilateral affection they lie on the diseased side in order to permit the free expansion to the healthy lung. Horses rarely lie down, cattle somewhat more often in the later course of the disease when they are much fatigued; lying down has, in consequence an unfavorable significance in horses. In severe cases sweating appears shortly before death.

The quantity of the urine is often diminished during the stage of hepatization, and in consequence the specific gravity is increased moderately; with the beginning of resolution the quantity increases for a period of one to three days, the urine becoming lighter in color and of lower specific gravity, even to below 1010 (urinary crisis). For diagnosis and prognosis, the diminution of the chlorine, during the stage of hepatization, is of importance; it is due probably to a diminished ingestion of food, but principally to the destruction and absorption of organic albumin into the circulation, where it takes up a portion of the sodium chloride in the blood plasma. The nitrogen and phosphorus elimination is increased, especially when the fever is about to go down and while the exudate is being absorbed

(Siedamgrotzky & Hofmeister. Wissinger. Storch) indicating an increased cleavage of protein—and of nuclein componnds in the total metabolism and in the exudate in the lungs.

The blood of horses with pneumonia shows a moderate hypoleucocytosis at the onset or during the fever; it changes into a hyperleucocytosis during resolution. The increase in the number of white blood corpuscles is mainly due to an increase in neutrophile also in acidophile cells (Sturhan, Wiendieck, Meier, Franke, Gasse). The number of red blood corpuscles and the amount of hemoglobin are diminished in most cases of pneumonia during the course of the disease; the values are, however, materially influenced by the ingestion of water and feed. If water is refused for several days and a loss of water of the organism occurs in consequence, the number of red blood corpuscles and the amount of hemoglobin may be relatively increased, though absolutely decreased (Wiendieck, Wetzl).

Deviations from the picture of typical and mild cases of pneumonia are not rare even in the absence of complications, but in such cases it is not possible to ascertain the cause of the atypical course. In stables with many horses one frequently notices that, simultaneously with a number of *typical cases of pneumonia, a few* cases occur with a markedly short duration. There is a sudden febrile attack with more or less severe general symptoms, but simultaneously with an improvement of the general condition the temperature goes back to normal on the second or third day (see Fig. 124). The thorax presents the signs of infiltration of the lungs (tympanitic, then dull sound, râles, sometimes indefinite breathing sounds), but they disappear after one to two days (pneumonia ephemera sive abortiva). Such cases are noted particularly in horses which have recovered from the disease in the past. In other cases the inflammation is localized exclusively in the central portions of the lungs which are not accessible to physical examination (Pneumonia centralis). A high, continuous fever persists for several days; the difficult respiration, saffron-yellow or rusty nasal discharge, as well as the typical fever curve suggest the presence of croupous pneumonia. In cases of this kind the inflammatory

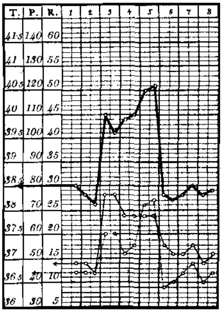

Fig. 124. Fever curve in croupous pneumonia of short duration in the horse.

infiltration may be discovered by means of the fluoroscope even in large animals.

Fairly frequent are those cases in which the inflammatory infiltration spreads from the pulmonary portions that are first affected to parts situated posteriorly and upward, or to the other lung (pn. progrediens). Under these circumstances the disease extends over a longer period, occasionally over several weeks, and the involvement of new portions of lung is indicated by elevations of temperature.

Recurrences occur exceptionally in cases in which the process has subsided, the fever disappeared and resolution taken place. In such cases the animal is, after a few days, again attacked by fever and the signs of pulmonary consolidation reappear at the place where they were found originally, or at another portion of the lungs (pn. recidivans).

Another deviation from the true type may be observed in cases where the inflammation is localized in unusual regions, sometimes restricted to the deeper portions of the lungs close to the hylus (pneumonia centralis). In the latter case, aside from a typical course of the fever, a diagnosis may be made only by the saffron-yellow nasal discharge which will probably be present, or by X-ray (sometimes also in large animals). The localization of the inflammatory process in the upper portions of the lung close to the vertebral column, or in the upper posterior region of the lung (see Fig. 121), is also atypical.

The symptoms of pneumonia may also vary according to the age and condition of the animal. In old, emaciated or weak animals a lower body temperature and more severe general symptoms (severe depression, loss of appetite, bad pulse) are observed (pneumonia asthenica or adynamica). The same symptoms occur also in very young animals, in which, however, a very high fever is simultaneously observed. Secondary pneumonia takes a course similar to the asthenic pneumonia and frequently leads to necrosis or heals very slowly. Ordinary pneumonia resulting from external influences shows a great tendency to take a slow course, in which case the fever is frequently less typical and under some circumstances (long-continued transportation) a great tendency to necrosis is apparent.

The symptoms of croupous pneumonia vary also according to different **species of animals.** Horses present the picture described above. Elevation of temperature does not occur as rapidly in cattle as it does in horses; the acceleration of respiration is, however, more marked in the former than in the latter. Cattle frequently present a weak, painful cough which can easily be brought on artificially. Saffron-yellow or rust-colored nasal discharge is absent, but one may observe a whitish mucoid nasal secretion. Bronchial breathing is heard more rarely than in horses; vesicular breathing over the affected portions is

more commonly weak or completely absent. The duration of the disease is longer; it lasts from one to three weeks, until *resolution occurs in cases of favorable termination.*

In cats, particularly in cases of the disease observed by Gaetner and others, besides the phenomenon of the nasal catarrh (see page 539), there can be observed a cough, fever (39.5° to 41.6° C., 103° to 107° F.), dejected, morose behavior, later pronounced apathy, and local thoracic symptoms. The animals usually die in from 3 to 6 days; only seldom does the disease last for six weeks or longer, and then sometimes it terminates favorably.

The affected animals usually die within three to six days, although exceptionally the disease may be prolonged to three weeks and more, and may then pass into recovery.

In birds pneumonia produces malaise, want of appetite, accelerated and whistling breathing through the open mouth; also sensitiveness and increased warmth of the chest; cough with expectoration of viscid, grayish, occasionally bloody or saffron-yellow discharge.

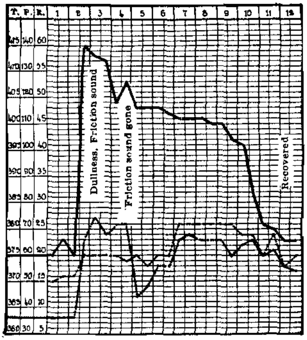

Fig. 125. **Fever curve in croupous pneumonia with co-existing fibrinous pleurisy in a horse.** Increased pulse frequency from the beginning of the disease.

Complications are comparatively frequent in the course of croupous pneumonia. Cardiac weakness manifests itself in a pounding apex beat and in a weak to filiform pulse which may be increased to double the normal number or more per minute. There is then also collapse, cyanosis, the veins are over-distended and a venous pulse is noted. These circulatory disturbances are caused partially by the weakening effect of bacterial toxins on the vessel walls.

Another very dangerous complication is suppuration or gangrene of the pulmonary tissue, particularly in horses. The advent of this complication is sometimes annonnced by a chill and by elevation of the temperature which may even have been falling; then there is continuous or remittent fever to the end. Disintegration of pulmonary tissue is announced nnmistakably by the onset of signs of cavity-formation (tympanitic sound persisting long in a circumscribed place, possibly associated with metallic sounds and amphoric breathing), and by the ap-

pearance of a disagreeable sweetish smell of the exhaled air. The symptoms of septicemia likewise become manifest. Under these conditions improvement occurs only very exceptionally. (Abscesses of the lung may break into a bronchus and may then heal completely.—Johne.)

Pleurisy is seen comparatively frequently; its occurrence as a complication is, however, quite variable. Tenderness of portions of the thoracic wall situated over the affected pulmonary areas, in connection with considerable acceleration of the pulse (Fig. 125), sometimes alone suggest the occurrence of a complicating pleurisy. Friction sounds are heard frequently or an abundant fluid exudate is formed in the thoracic cavity. The signs of pneumothorax (metallic sound, splashing, sudden respiratory difficulties) may occur in gangrene of the lungs. Fibrinous or serofibrinous pericarditis is observed more rarely and still more rarely acute verrucous endocarditis.

Icterus is seen frequently in horses and is usually due to a simultaneous gastro-intestinal catarrh or to involvement of the bile duct, or then to hemoglobinuria produced by the virus of the disease (Neubert). The icterus is apt to become marked, especially after a severe or a septic infection. Frequently a moderate and transitory albuminuria appears in consequence of parenchymatous degeneration or of acute parenchymatous nephritis occurring with the decline of the fever or rarely from the beginning, which increases gradually. Even in this case it is moderate (up to 0.2% of albumin), and usually disappears definitely with the fever. During resolution it may be associated with increased exfoliation of renal epithelia and excretion of leucocytes (Grossnickel). In contrast to Grossnickel's observation, the authors did not find that albuminnria is a constant accompaniment of croupous pneumonia in horses. Acute diffuse nephritis is rare, it leads to profound albuminuria and to the appearance of renal tube-casts; renal epithelia and occasionally also degenerated red blood corpuscles are seen.

In some cases urticaria or an acne-like eruption exists for a few days. Fröhner observed an exanthema similar to food rash. Among other complications tendovaginitis is met with (in 4.26% of the cases in horses, according to Schneider); it affects the distal tendon-sheaths of the flexors of the fore-feet, and produces an exudate that is free from microorganisms (Schneider). Less frequent are: acute tendovaginitis and arthritis of the extremities, laminitis in the horse, iritis, acute meningitis and encephalitis. In horses pneumonia is sometimes followed by purpura hemorrhagica.

Course. Croupous pneumonia is one of those diseases which run a very typical course. The stages of inflammatory congestion, hepatization and resolution follow each other reg-

ularly. In horses pneumonia usually reaches its climax toward the end of the first week, i. e., on the fifth to seventh day; then all morbid symptoms usually disappear rapidly within one day, sometimes a little more slowly, so that the duration is at the utmost two weeks in all. The period of convalescence corresponds to the intensity of the attack and usually leads to complete recovery.

Deviations from the normal course occur especially in cattle and swine, although they are not rare in horses. The sequence of the various stages remains the same, but the whole course may be of shorter or on the contrary of longer duration. Pneumonias caused by bacillus bipolaris or by external influences rarely take a typical course and those caused by the bacillus named (in cattle, hogs and sheep) may lead, within *a few days, to a fatal termination. Complications* which may develop also produce more or less marked deviations from the typical course and sometimes stand so much in the foreground that the clinical picture of pneumonia becomes completely clouded.

A fatal issue in consequence of suffocation may occur in very severe cases with extensive consolidation. Another cause of death may be grave degeneration of the myocardium. Cases with very severe infection lead to early and rapidly increasing prostration; usually toward the end of the stage of hepatization, the respiration becomes much accelerated and very forced, the heart is rapid and thumping, the pulse small, filiform, finally imperceptible; the mucosae become livid; there is finally a general perspiration and the animals fall to the floor. In the meantime symptoms of pulmonary edema have developed. Supparation and gangrene lead to death in other cases, after pyemic and septicemic symptoms have made their appearance. An edema of the glottis may sometimes produce death by suffocation.

A considerable number of cases of pneumonia is followed by a serous pleurisy; this must be suspected if dullness persists obstinately or even increases in the lower portions of the thorax and if more or less remittent fever is present. Severe pleurisy may cause adhesions of the pleura and this may lead to an asthmatic condition.

Chronic induration of the lungs not infrequently persists. *This is indicated by a long drawn out stage of resolution, some* difficulties of respiration persist; there may be moderate fever and the dullness does not clear up completely. A similar chronic process is sometimes developed in the neighborhood of a gangrenous focus, which may, even after weeks, produce an acute inflammation and cause death. A chronic pulmonary induration following upon acute pnenmonic symptoms appears in horses and cattle tolerably frequently only in certain years, while in other years pneumonia rarely leads to this complication.

The physical changes pointing to induration of the lungs disappear anyhow in a considerable number of cases 3 to 4 weeks after the fever has disappeared and the animals may be considered completely cured from a clinical standpoint.

In 0.4-2.8% of the cases occurring in the Prussian army the animals which had recovered presented after a few weeks the symptoms of paralysis of the larynx (q. v.). (Twenty-four [77%] of thirty-one thoroughbred horses of a stud, which had suffered from pneumonia, subsequently developed paralysis of the recurrent nerve [Plósz]).

Diagnosis. As a rule the diagnosis of croupous pneumonia is not difficult. In typical cases the sudden onset of high fever, which is continuous for several days and finally drops suddenly or by degrees, the regularly changing phenomena of auscultation and percussion, and the usual recovery in the second week, form a clinical picture that can hardly be mistaken for anything else. None of these symptoms, when present alone, is pathognomonic, not even the rust-colored or saffron-colored nasal discharge, which, moreover, is frequently lacking in horses. If, temporarily, high fever alone is present, the diagnosis of an impending croupous pneumonia can be made with great probability only in an infected barn. In view of this fact it is advisable to take regularly the temperature of all animals in the barn so that new cases may be recognized at the earliest possible time. Atypical cases of croupous pneumonia can be diagnosed from the peculiar changes in percussion and respiratory sounds which soon develop over relatively large portions of the thorax and moreover from the frequently atypical fever without previous or simultaneous bronchitis. In practice the very important differentiation between the various forms of specific pneumonia and the ordinary forms will be quite easy in the majority of cases if one takes into consideration the species of the animal, the color of the conjunctiva, the probably early or simultaneous development of a pleuritis, the presence of symptoms of other diseases (see the respective chapters in Volume I), and the probability of introduction from other sources. Sometimes a definite diagnosis can not be made, in which case it is advisable, in spite of the uncertainty of a specific diagnosis, to inaugurate effective treatment against specific pneumonia.

Errors of diagnosis may occur in pneumonia during the stage of hepatization, if a diagnosis has to be made on the basis of a single examination or if the disease is complicated by concomitant affections. One must think in this respect of pleuritis with effusion. Contrary to what is found in pneumonia, dullness is, in pleurisy, always in the lowest portions of the thorax, and in horses it is usually on both sides, the upper boundary being almost without exception a horizontal line; the resistance in the area of dullness is increased, breathing sounds cannot be heard at all or only along the upper boundary; the disease

comes on slowly, fever is usually less high, the course of fever irregular, breathing first of the abdominal type and later on very forced; dullness decreases or disappears after animal has been laid down and vesicular breathing takes its place; in smaller animals the dullness changes with changes in position in such a manner that it always appears in the lowest place. When pleurisy and pneumonia are present simultaneously some difficulty as to differential diagnosis prevails because the consolidated lung as well as the pleural exudate both cause dullness, while breathing sounds are often absent over the consolidated portions of the lungs. Edematous infiltrations formed on the lower thorax or lower abdomen may give some clue to the existence of pleurisy along with pneumonia. In doubtful cases an exploratory puncture should be made with a hypodermic syringe. The procedure is perfectly harmless even in the presence of an exclusive pneumonia. Only a positive result of the exploratory puncture can be utilized for diagnosis, because puncture may be negative even in the presence of pleuritic effusion.

Catarrhal pneumonia may furnish similar physical findings as croupous pneumonia if a number of small catarrhal foci have become confluent and have formed one larger consolidated area. However, bronchial pneumonia usually follows an extensive bronchial catarrh; it progresses slowly and lacks a typical course. One should never forget that adult horses usually suffer from croupous pneumonia, young foals and dogs exclusively, or at least preferably, from catarrhal pneumonia. The differential diagnosis from contagious pleuropneumonia in cattle is very important. During the stage of hepatization the findings may be identical in the two diseases; hence a reliable diagnosis can then only be made under consideration of the course. Contagious pleuropneumonia comes on gradually in contradistinction to the sudden onset and cyclical course of pneumonia, and cases with a comparatively rapid course last several weeks. The possibility that the contagious disease may be imported must always be considered. Inflammatory edema at the entrance of the larynx or in other parts of the body in the same or in other animals, the simultaneous occurrence of *enteritis and the rapid fatal termination,* point to a septic origin of pneumonia.

Foreign body pneumonia may be confounded with croupous pneumonia. It is, however, usually distinguished by a slower development. Fever is often absent at the onset or is very insignificant, bronchial breathing is likewise missed. After the entrance of foreign bodies from the stomach into the lungs, changes of the percussion sounds and respiratory sounds are noticed at first in the region of the diaphragm, and disturbances of digestion generally precede the affection of the lung.

In animals which are examined only after the fall in temperature, chronic inflammatory process, tumors, etc., must

be excluded on the basis of a polyuria present, or of anamnestic data, and with the aid of an observation extending over several days.

In horses tympanitic sounds in the neighborhood of the lower pulmonary boundary may also be referable to the colon; this can, however, be recognized easily because a tympanitic sound of the same pitch can be elicited also beyond the pulmonary boundary over the abdominal wall or even below the costal arch.

Prognosis. The stronger the animal has been before the pneumonic attack came on and the more the latter conforms to the classical type the better is usually the prognosis. The course of the fever is of the greatest prognostic importance. If the temperature curve falls rapidly or gradually after a continuous fever of several days, a typical—that is, a favorable—course may be expected. Fever that has lasted over a week and has perhaps risen above 41° C. causes anxiety. A continuous, though not high fever, during or after the stage of resolution, points to retarded or incomplete absorption of the exudate or to the development of a secondary pleurisy.

The behavior of the pulse is of special importance. If, in the absence of a complicating pleurisy the number of pulse beats has risen to twice the normal, the pulse being at the same time weak, the prognosis is fairly unfavorable. It is also necessary to consider the extent of the inflammatory infiltration; the chances for recovery decrease in direct proportion to the size of the consolidated territory. Hence, bilateral pneumonia is more serious than a unilateral affection. Deviations from the usual localization have a similar unfavorable prognostic significance. Slight albuminuria without cellular renal constituents in the urinary sediment indicates a severe infection but is of no importance otherwise. Central pneumonia and the involvement of the upper parts near the vertebral column show a higher mortality.

The age and nutrition of the sick animals must be considered. In very old or debilitated animals a slowly developing pneumonia is observed, with moderate elevation of temperature, yet with marked prostration (so-called asthenic or adynamic type), which is always very unfavorable in nature and which generally takes a fatal issue.

Every complication diminishes the chances for recovery more or less; particularly unfavorable are: pleurisy with abundant effusion, pericarditis, diffuse nephritis; to a lesser degree fibrinous pleurisy or intestinal catarrh. Symptoms of gangrene or suppuration of the pulmonary tissue point almost with certainty to an early lethal issue.

It must finally be considered that sequelæ may come on which will materially reduce the value of the animal. Since the advent of such sequelæ cannot be foreseen in the milder

cases, the prognosis must always be guarded until the lung affection has entirely disappeared.

The mortality varies in horses between 0-20%; it is usually between 10-15%; in milder enzootics much less. In cattle the mortality varies considerably more; sometimes all cases get well (Strerath, Coulon & Olivier, Gotteswinter); at other times a mortality of 40% has been observed (Krüger) and even more (Guillebeau & Hess).

Treatment. The establishment of the most favorable hygienic conditions, especially sufficient ventilation of the barn or sojourn in the open, is of the greatest importance in influencing the course and termination of the disease. Working animals must at once be taken off work, to be kept in a moderately cool, well ventilated place or in the open air during favorable weather, but they must be protected against rain and wind. Where there are larger numbers of horses, turning them out into a yard will influence those that are affected favorably and prevent the spread of the disease. Weak large animals, especially horses, should be placed in slings. They should be permitted to lie down only at intervals and not more than two hours at a time. Rubbing of the body is beneficial and invigorating to the patients.

Since in acute febrile diseases the animals usually eat only juicy, dainty feed, herbivora should have fresh green feed, or if this cannot be had, good fresh hay, bran or flour mash perhaps mixed with some grains, also roots, beets, carrots, etc. If sick horses appear to prefer oats they should have them, mixed however with chopped vegetable roots. Carnivora should receive fresh milk, finely chopped boiled or roast meat, also strong meat-soups with one or more eggs. All food should be given in small but often repeated rations. The animals should frequently be offered fresh, but not too cold, water, also during the night, since the animals, and particularly the horses, are too weak to get to the suspended or otherwise placed water vessels. It is also advisable to clean the mouths of the sick animals with pure water before offering them food. They often take it then even if they appear previously to have no appetite. Where aspiration is threatening on account of disturbances of deglutition, food and water may be withdrawn, and moderately or well nourished animals may be made to fast for a few days. Later on, however, these patients and those which are weak or emaciated must be nourished artificially (see page 43). The same procedure must be instituted with patients who entirely refuse to eat. Where water by mouth has to be withheld completely, water injeetions per rectum must be resorted to.

Drugs can be dispensed with in all cases with a typical course. If the pulse varies only slightly from the normal, alcohol is indicated in small, often repeated doses (ordinary

spirits), larger animals 25-50 gm., sheep, goats, swine 10-20 gm., carnivora 1-2 gm.; wine ½ quart down to teaspoon doses; cognac, carnivora ½ teaspoon. Sulphuric ether (10 gm., 5 gm., or 0.25-1.0 pro dosi) may likewise be administered. If the number of pulse beats is increased to more than twice the rate, and also if they are weak, cardio- and vasotonic drugs are indicated. The most serviceable of these drugs is camphor, as ol. camph. according to Fröhner in tolerably large doses (for large animals 20-50 gm., for smaller animals 4-10 gm., for carnivora, 1-2 subcutaneously in one dose). The same results may be had with the much cheaper ol. camph. syntheticum (Fröhner) used in similar doses. Fischer saw very rapid and favorable effects following the intravenous infusion of liquor camphoratus physiologicus (to 1 liter of sterile physiological salt solution add spirits of camphor drop by drop while stirring constantly until the camphor begins to precipitate, then filter), of which he administered 1 liter at body temperature to horses. The slight excitement which follows is without significance. Schmitt-Jensen administers 1.2 gm. pulverized camphor in 1 liter of physiological salt solution or Ringer solution, which he heats to 75° C. (167° F.) in a wide-bottomed container on a water bath, shakes it thoroughly and then allows it to cool gradually on the water bath to prevent the partial precipitation of the camphor. Sterilized watery solutions of camphor can also be purchased and administered with good results in doses of 200 to 300 cc. (Rips, Brachmann).

Caffeine (for large animals 6-8 gm., for small animals 0.5 to 1 gm., subcutaneously every six hours) is likewise beneficial. (Gmeiner claims to have shortened the course of cases of pneumonia by the use of subcutaneous injections of caffeine.) Strophantinum purissimum Merck (horses 0.003 gm.) recommended by Regenbogen by subcutaneous injections in cardiac affections has not proved beneficial in the authors' cases. It acts much better by intravenous injection (for horses up to 0.015 gm.—Dorn, authors' own observation). For the purpose of subcutaneous injection strophantine must be dissolved in enough water (6-10 gm.) in order to avoid necrosis of the skin (Fröhner); however, even if applied in this dilution, a painful infiltration at the place of injection, which lasts several days, cannot be avoided. Such swellings are likewise frequently seen after injections of caffeine. Digitalis preparations to be used are: pulverized leaves of digitalis (for large animals 2-5 gm., for smaller animals 0.5-1.0 gm., for carnivora 0.05-0.3 for three to four days); also folia digitalis dialysata (for larger animals 5-15 cc. per os); further, digitalysatum Bürger (small animals 20-30 drops, subcutaneously). According to the investigations of Salvisberg, digitalis preparations are destroyed in the stomach of ruminants; hence the proper preparations must be given subcutaneously or intravenously.

It must not be forgotten that digitalis has a tendency to produce *abortion in pregnant cows*. The general use of the excellent preparation digalen (horses 5-15 cc., cattle 20-30 cc., subcutaneously or intravenously) is frequently prohibited by its high price (Dorn); tincture of strophantus has been found unreliable (Regenbogen, Gmeiner).

In the face of threatening cardiac weakness the proper preparations of digitalis must be given intravenously because then only can they produce the desired effect within a short time.

Croupous pneumonia is not a very favorable disease for the use of preparations of digitalis and strophanthus, since the disturbance in the circulation as a rule is caused exclusively or predominantly by a general decrease in the tonus of the arteries, in which case drugs that constrict the blood vessels or the administration of large quantities of fluid are much more effective. Remedies suggested are camphor and caffeine, which act especially in this manner: barium chloride (2 to 3 gm. internally, 0.1 to 0.15 gm. intravenously for large animals); upon the suggestion of Wirth, repeated subcutaneous injections of adrenalin, tonogen, suprarenin, paranephrin (for doses see page 166); intravenous infusions of physiological salt solution or Ringer solution at body temperature (for horses 4 to 6 liters), and artificial serum (same dose). In case of weakness of the heart (cyanosis of the mucous membranes, injected veins, accelerated or irregular pulse) the use of drugs prepared from the suprarenal glands or intravenous infusions of large quantities of fluid should not be attempted in order to prevent fatal edema of the lungs or collapse.

Venesection, which was formerly practiced universally, may bring temporary relief in the beginning; it does not, however, influence the course of the disease at all, and is by no means devoid of danger, on account of decrease of the tonicity of the arteries.

Fever does not, as a rule, require any special treatment, except in the presence of hyperpyrexia, when cold sponging, or irrigation or injections of cold water into the rectum may be indicated. However, cold douches should not be used during the cold season nor in very young or very debilitated animals. Of antipyretics the following may be used: antifebrin (15-30 gm. for large, 1-4 gm. for smaller animals, 0.1 to 1.0 gm. for carnivora pro dosi); phenacetin (the same doses) or antipyrin (the same doses). Lactophenin, which is much higher in price, may be given to smaller animals (0.5-1.0 pro dosi). With the exception of phenacetin or lactophenin these drugs must be used cautiously, since they may bring about collapse. The salicylates are indicated much less (acid. salicylicum, sodium salicylicum, salipyrin, salol, aspirin, dymal) since they often produce intestinal disturbances or irritate the kidneys.

In influenzal pneumonia of horses intravenous injections of salvarsan or neosalvarsan give good results. Good results are sometimes also observed following the administration of arsalyt, arsinosolvin, atoxyl, arsanil, and metarsan (for particulars see volume 1). In man, Grund observed immediate disappearance of fever and a rapid recovery following a single intravenous injection of collargol (about 10 cc. of a 1 per cent. solution) and the internal administration of quinin repeated several times (0.5 to 0.3 gm. twice daily). Cahn-Brenner had the same results without the previous administration of collargol if the quinin was given in the early stages of the diseases in doses of 0.5 gm. for two days and again after an interval of 24 hours if necessary. Best results were obtained with intramuscular injections (0.5 gm. quinin hydrochloride and 0.25 gm. urethane in 5 cc. distilled water, or chininum dihydrochloricum carbamidatum Merck).

Of bactericidal solutions, Houdemer used jodokol (5 cc. daily intravenously for 5 days) and Fournier a 1% creosote solution in 60% alcohol (10 cc. twice daily intravenously). Tallianines (10 cc. intravenously for the horse) were used with very little success. Beckhard and Schneider recommend the much cheaper ozonal (ozonized liquid turpentine) in 10 cc. doses (intravenously). Bouchet injected hydrogen peroxide (12% by volume) intravenously in doses of 20 to 90 cc. (sometimes followed immediately by dyspnea, repeated defecation, and slight colicky symptoms), which was followed by a permanent drop in body temperature. In suitable cases this drug probably acts favorably by exciting a leucocytosis and by influencing the colloidal condition of the body fluids. In man the administration of optochin (0.5 gm. intravenously, followed by 0.25 gm. per os) perceptibly shortened the duration of the disease, but was now and then followed by blindness. A similar favorable action was observed by Schern in eight horses after the internal administration of optochin (4 to 6 gm. per 100 body weight) as a bolus.

The intravenous injection of typhoid or cholera vaccine (5 to 10 cc.), recommended by Besskó and Szily for the treatment of croupous pneumonia in horses, is essentially a protein therapy (parenteral administration of protein foreign to the species). Although it often brings about a rapid drop in the body temperature to normal, it is frequently followed by a severe intoxication (in the author's case, fatal in three hours). A similar action, but free from danger, may be obtained by subcutaneous (or, better, intravenous) injection of foreign blood serum (in quantities used in the administration of immune serum, or two to three times that amount). The artificial and more expensive protein preparations, such as laktosan, serocasein, caseosan (sterile solution of casein), aolan, proteosan, ergolactin, ergo-

tropin, eugalaktan and others may be used in a similar manner. (The parenteral administration of incompletely sterilized preparations may be followed by fever, and upon reinjection may result in anaphylaxis after a lapse of a certain time, or may be followed by necrosis or purulent swellings at the point of injection). The subcutaneous injection of sterilized milk, which has a similar action and is recommended in other infectious diseases, was not suitable in the hands of the author, since it is prone to cause purulent swellings at the point of injection. The parenteral administration of foreign proteins on the one hand causes leucocytosis and on the other hand the formation of non-specific bactericidal and immunizing substances, and in this manner may, to a certain degree, have a favorable influence upon pneumonia. (Caution should be observed with respect to the possibility of toxic effects from the injection of foreign proteins. See serum sickness.)

Inhalation of oxygen, as recommended by Eberlein and Toepper, leads to improvement in the general condition and to a decrease of the dyspnea.

Delayed resorption may be stimulated by Priessnitz' applications and by diuretic drugs; of the latter we should use preferably acetate of sodium or potassium (25-30 gm. for large, 3-10 gm. for smaller animals, 0.2-1.0 gm. for carnivora, two to three times daily); diuretin, theozin, theophyllin (for carnivora daily three times 0.2-0.5 gm.); iodide of potash (for large animals 10-20 gm. pro die per os) iodipin (for large animals 30-50 gm. subcutaneously), fibrolysin (large animals 10 cc. every third day, dogs 2 cc. of the solution). The resorption of the exudate may be stimulated in protracted cases by puncture of the infiltrated lung with the hollow needle of an injection syringe; the procedure is similar to that used in exploratory puncture of the thoracic cavity.

The repeated inhalation of atomized disinfecting fluids (solution of corrosive sublimate 1:2000-4000, 2 to 3% solution of carbolic acid or creolin) may diminish putrefactive processes in the air passages, but they cannot prevent putrefaction in the lung tissue itself after pulmonary gangrene has set in. Intratracheal injections of a 1% solution of carbolic acid (for horses 60-100 gm. at one time) or of a creosote or formol solution (see page 611) are better adapted for the treatment of pulmonary gangrene.

When pleuritis is present, it calls for special treatment (q. v.)

Stomachics (*rheum, root of calamus, gentian, or their tinctures for smaller animals*), artificial Carlsbad salt, hydrochloric acid with pepsin may be administered to stimulate appetite. Convalescent animals must be well nourished and not used for work until they have regained their full strength. Moderate exercise in the open is beneficial if the weather is favorable.

Since croupous pneumonia is usually a localization of some

acute infectious disease, such prophylactic measures as are recommended for the particular infection, must be carried out.

6. Catarrhal Pneumonia. Pneumonia catarrhalis.

(*Bronchopneumonia, Pneumonia lobularis s. insularis.*)

Catarrhal pneumonia is an inflammation of individual lobules or groups of lobules of the lungs, leading to an occlusion of the alveoli with desquamated epithelia and a cellular exudate, which usually develops by the continuation of a bronchial catarrh into the corresponding lobules or simultaneously with bronchiolitis.

Etiology. Those factors which may cause bronchitis or bronchiolitis (capillary bronchitis, see page 586) may all cause catarrhal pneumonia, but there are special factors which are particularly prone to produce bronchopneumonia. The inhalation of very hot air, or air containing irritating gases, the entrance of foreign bodies (fluids or solids) into the air passages may cause bronchopneumonia, which may frequently terminate in gangrene, particularly if caused by foreign bodies. Foreign bodies generally get into the air passages by aspiration in swallowing, but they may also be inhaled in regular respiration. Wool threads (Dralle) and parts of nut cakes (Habicht) may in this manner be inhaled into the lungs of cattle, and if these animals are fed with cottonseed or ground-nut cakes there may be numerous cases of this kind. Bronchopneumonia seen in horses after chloroform narcosis also occurs in consequence of aspiration, if these animals receive solid food directly after the narcosis (Ducasse). In calves foreign-body pneumonia may develop after the forcible pouring in of liquid foodstuffs for the purpose of increasing the weight (see page 699). The observations of Siedamgrotzky and Johne concerning an enzootic bronchopneumonia of cattle in the neighborhood of Freiberg are highly interesting. This form of pneumonia developed in consequence of the inhalation of smelter-smoke containing arsenic and it predisposes very much to the development of tuberculosis.

Parasitic worms (see page 595) are frequently, moulds rarely, the cause of catarrhal pneumonia.

The great majority of cases of catarrhal pneumonia develop on the basis of an infection. The same organisms which cause croupous pneumonia may especially be active (Weichselbaum), particularly in very young, or on the contrary, in old debilitated animals.

The bacillus bipolaris septicus may cause bronchopneumonia, particularly in cattle. (According to Smith, the sporadic pneumonia of cattle is a bronchopneumonia caused by the bacillus bipolaris). Bronchopneumonias are also often caused

by bacillus pyogenes. However, this bacillus probably invades the lungs only secondarily and is able to display pathogenic *properties there only if the bronchial mucosa has become predisposed* at the time of the invasion or previous to it (Holth, Reisinger) in consequence of non-specific irritation (cold, inhalation of dust, congestion of the lungs). Martens, Berstel, Schmidt and Lewek have described a bronchopneumonia occurring in Germany, either sporadically or enzootically, as an infectious disease sui generis, beginning with the symptoms of catarrh of the upper respiratory passages (see page 574) and leading to bronchopneumonia in a portion of the cases. It can not yet be decided what is the relation between this affection and bronchopneumonia caused by the bacillus pyogenes, which was described more fully by Reisinger. Lewek found Gram-positive bacilli or Gram-positive cocci or both in the affected lungs. Prolonged transportation on railroad cars or on board ship, unhygienic conditions and colds play a predisposing rôle. The disease is *particularly common in market cattle.* In cows Miessner & Bartels observed a stable enzootic of bronchopneumonia with necrosis and formation of sequestra; at the same time, necrosis of the liver and catarrhal mastitis. The necrosis *bacilli were found in the necrotic foci and in the milk;* they had probably localized secondarily in the bronchopneumonic foci. Enzootic bronchitis in fattened cattle, described by Koudelka, is occasionally associated with bronchopneumonia.

Bronchopneumonia caused by the bacillus pyogenes occurs also in hogs (Borges).

Murray observed an epizootic bronchopneumonia in the *Mississippi Valley in North America,* which, although causing only 1% losses by death, nevertheless wrought great damage from a falling off in condition of the animals as a result of depression of the appetite lasting from 7 to 10 days. He isolated from the animals a Gram-negative micrococcus, which, upon intravenous injection of a small amount of a culture, produced symptoms identical with those following natural infection.

The causative organism of an infectious bronchopneumonia observed by Roebert and Deich and more recently by Kuhn in dogs of all ages is still unknown. In streptomycosis of cats, de Jong found bronchopneumonic foci only once.

In rabbits the disease develops frequently in the course of infectious nasal catarrh, but may also occur as a primary lesion in the latter disease (so-called "Brustseuche" of rabbits). The colon-like bacillus described by Suedmersen also produces an enzootic *bronchopneumonia* and a *catarrhal* condition of the air passages.

A fatal pneumonia in young fowls following a catarrh of the upper air passages was observed by Eber in a year during which influenza was present, also otherwise during the spring and summer months, particularly during the past war. Pneumonia was

also observed by him following inflammation of the oviduct, the resulting peritonitis spreading to the lungs.

Bronchopneumonia often occurs secondarily, especially in young animals, in the course of acute infectious diseases. For instance in distemper in dogs, in hemorrhagic septicemia in sheep which was also known as sheep glanders, in some cases of malignant catarrhal fever, in foot-and-mouth disease, in sheep-pox, purpura hemorrhagica, hog cholera; more rarely in chicken cholera. Tubercle and glanders bacilli, also ray-fungi may be the cause of catarrhal pneumonia. (For chronic catarrhal pneumonia occurring in South Africa and there called "Jagziekte," or "Hartslagziekte," see page 640.)

Predisposition. Predisposing factors often play an important rôle. Such predisposing factors are: very early, or on *the contrary, very advanced age, cold, debility of the organism as it occurs in anemia, in grave nutritive disturbances, in rachitis, etc. These predisposing causes may, however, also be absent in infectious catarrhal pneumonias.*

Pathogenesis. Infection takes place primarily through the air passages, although in some cases, particularly in specific infectious diseases, an infection from the blood stream cannot be denied. For some unknown reason the virus seems to multiply, especially in the contents and mucosa of the air passages, while it spreads only slowly or not at all along the connective-tissue septæ. Accordingly, the inflammatory process spreads especially along the bronchi to the alveoli contiguous to the infected bronchi. Undoubtedly there are exceptions to this mode of development, since some infectious agents, as in croupous pneumonia, rapidly penetrate along the spaces in the connective-tissue septæ, thus producing inflammatory processes of a large area. The formation of a noncoagulating exudate, consisting predominantly of desquamated epithelium and white blood corpuscles, much mucus and serum of a low albumen content, is probably due to the exclusive or predominant multiplication of the infected agents in the contents of the bronchi and alveoli. In some forms of the disease the exudate may have an admixture of more or less fibrin, in case the micro-organisms are especially virulent or have rapidly spread into the tissue spaces and have thus more or less affected the walls of the capillaries.

This disease is distinguished from croupous pneumonia, as the micro-organisms multiply exclusively or predominantly in the air passages and are there protected by foreign bodies or otherwise, and are more seldom and to a lesser degree subjected to the lytic action of the body fluids, especially since their location in the body does not tend to promote the formation of immune bodies either locally or in the body as a whole. For this reason the disappearance of the fever and the resolution so characteristic of croupous pneumonia is absent.

The influence upon the respiration and circulation is the

same as in croupous pneumonia (see page 652). In continued disturbance of respiration, a greater significance must be attached to the bronchitis.

Anatomical Changes. The inflammation is, as a rule, confined to certain foci, to individual lobules or groups of lobules; hence there are at first smaller, later on larger pneumonic areas, which may become confluent and form large consolidated territories. These consolidated areas are usually in the neighborhood of the lower and anterior pulmonary margin. They are solid; not as firm, however, as in croupous pneumonia. The cut surface is at first flesh-colored, later on more and more grayish-red, and, when the course of the affection is very slow, yellowish or grayish-white and then dry. The cut surface is not *granular; aside from catarrhal, there are also seen atelectatic* foci, which are a little darker, but airless, wedge-shaped, sunken in under the general surface and uniformly meat-like to the touch. Other portions are not uncommonly emphysematous and bloated. In cattle interstitial pulmonary emphysema frequently exists simultaneously. If the disease has lasted a long time one may see whitish or greenish purulent foci of various sizes in the consolidated areas, and in these Preisz' pseudotubercle bacillus, or bacillus necrophorus, in hogs and cattle the bacillus pyogenes are found occasionally. In the course of pneumoparatyphoid in hogs, the reddish-gray, slightly moist pneumonic areas contain hazelnut to walnut-sized caseous foci which at first are reddish-white and lardaceous, then grayish-yellow to greenish-gray; the center is occasionally softened. The caseation is rarely diffused. Similar cheesy foci are found in the bronchial lymph nodes; there are usually cheesy changes in the intestines (Glaesser).

The bronchi, especially the smaller ones, always contain an abundant mucoid or purulent, occasionally more caseous exudate, which is discharged from the cut surface. The mucosa is reddened, loosened and swollen.

In consequence of serous and cellular infiltrations the interlobular connective tissue appears occasionally gelatinous or whitish; this is often the case in cattle, occasionally in hogs, and only exceptionally in other animals. The pleura frequently appears healthy, occasionally somewhat roughened, at times also covered by a fine fibrinous membrane. The bronchial lymph glands are in a condition of acute swelling.

The microscopical examination of the bronchopneumonic focus shows dilatation of the blood capillaries, serous and round cell infiltration of the intraalveolar connective tissue; copious exfoliation of the fatty degenerated alveolar epithelia and, at times, polynuclear giant cells; numerous leucocytes and few red blood cells in the alveoli and occasionally fibrin-threads which are frequent in cattle.

Symptoms. Catarrhal pneumonia is usually preceded by the symptoms of bronchitis or bronchiolitis; frequently the former manifests itself simultaneously with the latter. In the

former case an aggravation of the symptoms points to the advent of catarrhal pneumonia.

When bronchiolitis is already present, an elevation of the temperature above 40° C. at once creates the suspicion that catarrhal pneumonia has developed following the primary disease. This may be held with almost absolute certainty if the patient is very young or very old. The fever does not show a typical course; it is of a remittent type and disappears gradually after recovery has set in (Figs. 127 and 128). Catarrhal pneumonia of adult animals may, for some time or even up to the end, run its course without fever and without other grave general symptoms.

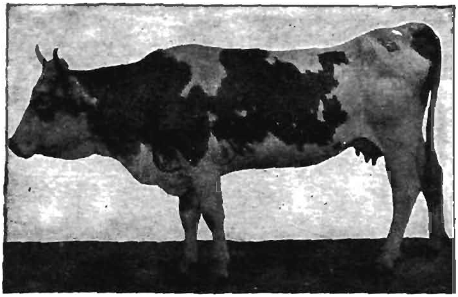

Fig. 126. Bronchopneumonia and tuberculosis of the lung. a, prescapular; b, thoracic dullness.

The respiration is accelerated and made difficult in direct proportion to the extent of the inflammatory process; dogs, more rarely other animals, blow up their cheeks with every expiratory movement (so-called labial breathing). The animals cough from time to time or continually; the cough is usually short, dull, frequently painful, and consequently the patients try to suppress it. One usually observes also an abundant nasal discharge.

Percussion produces pain and frequently excites cough. Careful, attentive percussion reveals dull or very low sounds in circumscribed places of the thorax. These changes are usually found above and along the lower pulmonary border, only exceptionally in the middle or upper third of the thorax. The percussion sound may have a tympanitic timbre at the boundary of the area of dullness or in other places. Small animals sometimes present the cracked pot sounds.

Ausculation reveals a variety of catarrhal sounds, sometimes in circumscribed places, as a rule, below, at times over, the whole of the thorax. Sounding (metallic) râles have a

special significance if they are present. In exceptional cases, if the lungs have become consolidated to a larger extent, while the lumina of the larger bronchi have remained open, one hears bronchial breathing in the area of dullness; as a rule, however, the breathing sounds are more or less weakened, because the bronchi are stretched by the catarrhal secretion.

The pulse is accelerated, at first quite strong; later on it becomes weakened.

The general condition suffers materially in most animals, both on account of the fever, and on account of the respiratory difficulties and the tormenting attacks of cough. Small animals usually rest on the sternum. The appetite is diminished or entirely suppressed; sucking animals do not go to the mother.

Infectious bronchopneumonia of cattle follows, as stated, after infectious catarrh of the upper respiratory passages (according to Schmidt, in 50% of the cases). Three to four days elapse, according to Lewek, before catarrhal pneumonia develops; catarrh of the upper respiratory passages may, however, assume a slow, insidious course and may bring about pneumonia at any time (Reisinger). If this is the case the fever comes on suddenly, and dullness and bronchial breathing can soon be demonstrated over the anterior and lower portions of

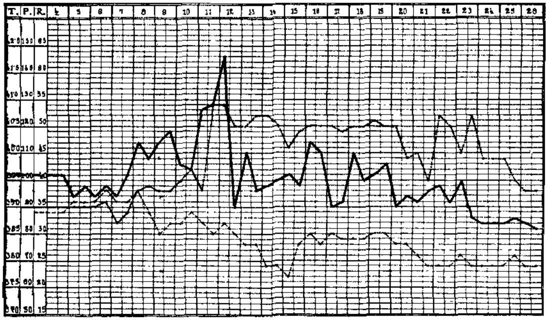

Fig. 127. Fever curve in catarrhal pneumonia of the dog, terminating in recovery.

the thorax. The fever may, however, disappear again within a short time; the appetite may improve, yet the signs of pneumonic consolidation persist without influencing the general nutrition or the yield in milk. In this form the disease lasts about fourteen to eighteen days and reaches its climax about the tenth day; then the symptoms decrease and most animals recover completely (Lewek). Occasionally the course may be less favorable. In both forms interstitial pulmonary emphysema is not infrequently developed.

In the cases of infectious bronchopneumonia in dogs observed by Kuhn, symptoms of pleuropneumonia were present. In 60% of the cases the animals died within a few days or only after

2 to 4 weeks. Convalescence of the remaining animals required several weeks, but recovery was then complete. The enzootic was brought to a halt by the complete isolation of the healthy dogs.

The bronchopneumonia observed in the course of chronic septicemia in rabbits (chronic "Brustseuche") manifests itself, according to Sustmann, in depression, coughing, sneezing, emaciation, changing appetite, yellowish-white nasal discharge, purulent conjunctivitis, light dyspnea, and sometimes diarrhea. Treatment was not successful.

Course. Catarrhal pneumonia follows a very variable course, in accordance with the variety of its causes. It develops very rapidly in some cases and may have assumed a considerable extent within a few days; the development is slower in other cases and the course may extend over several weeks and even months. The fever subsides in favorable cases toward the end of the second or third week, or often even earlier; the cough becomes easier, moist, less frequent; the respiration less accelerated and forced, and complete recovery takes place. Renewed elevations of temperature may come on during the period of recovery; this is then due to the formation of new inflammatory foci.

In other cases the disease takes a fatal termination, and the animals die from suffocation, cardiac paralysis, exhaustion, intercurrent pleuritis or pericarditis, or from profuse diarrhea, occasionally also from pulmonary gangrene or in consequence of a general sepsis. Again, in other cases there may remain a chronic pulmonary induration; the animals then suffer in their nutrition and from respiratory difficulties. Young animals are retarded in development under these circumstances.

Diagnosis. The greatest difficulty is offered by the differential diagnosis between catarrhal pneumonia and bronchiolitis; a careful physical examination, however, often enables us to come to a definite conclusion. High fever, absence of vesicular breathing, and particularly dullness on percussion, speak in favor of catarrhal pneumonia. Catarrh of the bronchioles rarely exists for any length of time without the advent of catarrhal pneumonia. Croupous pneumonia can be distinguished by its much more rapid development, its acute and cyclical course, by the extensive and strong dullness, the bronchial breathing which is frequently present over a wide territory and finally by its often favorable termination. Bronchopneumonia in cattle can not be differentiated from pulmonary tuberculosis without any further study when it takes a somewhat protracted course. In contradistinction to pulmonary tuberculosis, bronchopneumonia does not lead to severe emaciation, even after severe local changes, and the catarrhal sounds remain, as a rule, confined to the anterior and inferior portions of the thorax.

Prognosis. The prognosis is the more unfavorable the younger or the older the animal. The poorer the state of nutrition, and the higher the fever, the slighter are the chances for complete recovery. The course is particularly unfavorable if the catarrhal pulmonary infection comes on during an acute infectious disease. Röbert observed a mortality of 30% in epizootic bronchopneumonia of dogs; Deich, 50%; Schmidt reported 30% mortality in contagious pneumonia of market cattle.

Treatment. The hygienic environment and diet should be regulated according to the same principles as were laid down for croupous pneumonia (see page 666). Sucklings suffering from lack of appetite must be fed with freshly drawn milk or with boiled milk cooled down to body temperature; two to four eggs may be added; eggs can also be given raw or mixed with warm wine soups.

To improve the bronchial catarrh inhalations may be practiced (see page 528); inhalation of oxygen might likewise be tried. Narcotics (see page 667) should be used only in the presence of tormenting, convulsive cough, and even then very cautiously. The regulation of the heart's action requires special attention; the principles are the same as those laid down for croupous pneumonia (see page 666). Priessnitz' applications to the thorax may be useful. If a complicating pleuritis, pericarditis or enteritis is present, they should be treated suitably.

Prophylaxis. If the disease is due to an infection, the animals which are still healthy should be removed to suitable quarters, preferably to some distant place; they ought to receive a change of diet and water and disinfection should be practiced repeatedly. Newly bought cattle should be isolated for one week (Lewek).

Infectious Bronchitis and Bronchopneumonia in Horses. This disease, also known as "Brussels disease," "Ghent disease," "horse typhoid," "remount disease," "horse typhoid of Flanders," "horse typhoid of Ghent," and "malignant strangles," occurred soon after the outbreak of the World War and spread rapidly through the army horses and those of the civilian population. A few cases had already been observed in past years (in 1912 Brunschwik in France recommended a special treatment for the disease), but at that time it was known under a different name. According to Kossmag, the disease known in Germany as "horse trader's strangles," "acclimatization fever," certain cases of infectious catarrh of the upper air passages, and the disease called "skalma" by Dieckerhoff, are closely related to this disease. French and Belgian veterinarians consider it as the pectoral form of typhoid fever with

bronchitis and pneumonia. The disease was accurately described by Wirth, Reinhardt, Boesch, Kossmag and others. A similar disease was observed by Zwicky in Switzerland among mules.

Etiology. The virus is still unknown. According to Kossmag, the virus belongs in the class of filtrable viruses and the disease is related to the influenza group of diseases and particularly to skalma. In the nasal discharges and the diseased lung tissue streptococci were regularly found almost in pure culture (Wirth, Reinhardt) or together with staphylococci and other bacteria, but their pathological significance could not be established (Boesch). Schroeder found bipolar organisms. The same is true of the peculiar trypanosomes found by Ruether in recent foci in the lungs, and of the very short spirochetes that were always found in the blood in cases of relapse. The disease is very contagious, rapidly spreads to all the horses in the barn in the form of a barn enzootic, and affects all horses regardless of age, breed, sex or condition. Newly bought animals and particularly young and very old horses, as well as the heavy breeds, are especially inclined to become infected. According to Boesch, only animals which have recovered from influenza are not affected by the disease.

Anatomical Changes. Pronounced emphysema of the lungs is present; the lungs are enlarged to two or three times their original size. Especially the apical and cardiac lobes are greatly ballooned, and the edges of the lungs appear like spools. Besides, there are found in the anterior portion of the lungs yellowish-brownish red or dirty dark-red foci of varying shape and hepatic consistency and which on cross-section appear moist, glistening and smooth and in some cases granular to nodular or marbled. The interlobular septæ are widened and of a gelatinous character. Purulent or gangrenous foci up to the size of a hen's egg and with ichorous content are frequently found. These may have coalesced into a single pus cavity. Large quantities of mucopurulent or clotted granular masses are found in the bronchi and frequently occlude them entirely. The bronchial mucous membrane is reddened, swollen or thickened. Bronchiectasis is frequently encountered. The bronchial lymph glands show edematous swelling. Wirth frequently found purulent retropharyngeal and subparotid lymph glands. Degeneration and septicemic changes are often found in the large organs.

Symptoms. In some of the cases the animals seem tired and partially exhausted, or general disturbances may still be absent. The conjunctiva is a very pronounced scarlet, frequently showing a yellowish tinge. Fever is always present from the beginning (39.5° to 40.4° C. [103° to 105° F.], sometimes 41° C. [106° F.] and more), is atypical (Fig. 128), and usually

decreases gradually after a few days, but certain variations remain (frequently a difference of several degrees in the course of 2 to 3 days), or afebrile periods may be followed by another rise. The pulse varies between 48 and 60; only when the condition has become aggravated will it rise to between 60 and 80. The pulse, however, remains pretty strong unless septicemia develops as a result of gangrene of the lungs.

A profuse nasal discharge is always present. At first it is mucoid or mucopurulent; soon becomes purulent, and then it comes away in large creamy or dirty yellowish-green masses which later become foul-smelling. In the majority of cases there is also a seromucoid, exceptionally a mucopurulent, discharge from the eyes. Furthermore, frequent coughing is ob-

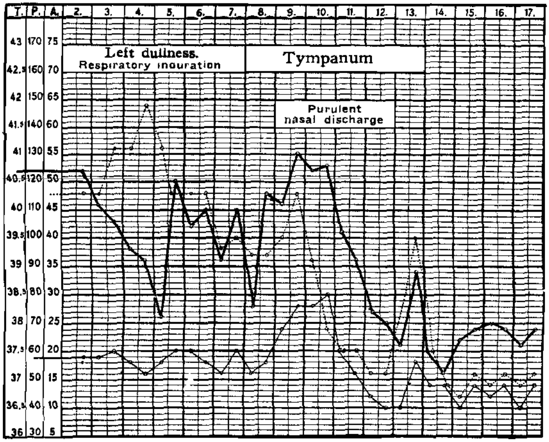

Fig. 128. Fever curve of infectious bronchopneumonia of a horse, terminating in recovery.

served, at first strong, later often very weak. In the further course of the disease the submaxillary lymph glands frequently become somewhat swollen, but abscess formation is very seldom observed in these and other lymph glands of the head.

Respiration is only slightly accelerated and labored. In case pneumonia develops, it becomes greatly accelerated (40 to 60 per minute), and at the same time dyspnea also develops, predominantly inspiratory, with more or less even movement, and

with strong dilatation of the nostrils and shaking of the whole body. Also an emphysema may develop under the skin as the result of pulmonary emphysema.

Contrary to other observers, the author detected changes in the percussion sound similar to those observed in common bronchopneumonia (see page 675). Increased vesicular breathing and the occurrence of bronchial breathing in circumscribed areas have also been frequently reported by other observers. Rales are never absent.

Food and water are usually taken; in severe cases, however, the consumption is decreased or they are entirely refused. Now and then a temporary diarrhea, usually foul smelling, is observed.

As complications there arise frequently gangrene of the lungs, seldom pleuritis and cerebritis, more frequently purpura hemorrhagica, and sometimes founders.

Course. The course is usually slow. In very favorable cases a gradual and decided improvement can be observed after 2 to 3 weeks, sometimes even earlier, whereupon, in spite of a probable return of fever, recovery takes place after an illness of about 4 to 5 weeks. As a rule, however, the disease lasts from 7 to 8 weeks, or even 9 to 12 weeks. Sudden exacerbations or relapses, even with a fatal termination, sometimes occur, even after weeks of improvement or absence of fever. On the other hand, cases are met with which rapidly grow worse from the very beginning and terminate fatally in from 3 to 12 days. The mortality runs from 12% to 28% (Reinhardt, Boesch) up to 63% (Wirth). As a result of chronic bronchitis, atelectasis, induration or emphysema of the lungs, permanent heaves sometimes develop.

Diagnosis. Infectious bronchopneumonia can be distinguished from influenza by the peculiar yellowish coloration of the conjunctiva and the rust-colored nasal discharge; furthermore, by the atypical fever, the slight changes in the percussion and respiratory sounds, the irregular course, and by the fact that the fever does not subside after an injection of salvarsan. In contrast to strangles, abscess formation in the lymph glands occurs only seldom, in spite of the severe purulent catarrh. Epizootic pharyngo-tracheal catarrh is characterized by rapid spread and short duration, with a light catarrh of the respiratory passages. Influenza can be similarly differentiated from it. This disease, furthermore, shows a glassy swelling of the conjunctiva, feed is refused from the beginning, and pronounced intestinal disturbances are present. In infectious anemia, which has repeatedly been mistaken for bronchopneumonia, bronchopneumonic changes and, as a rule, cough are absent; while in the acute and subacute forms, which alone are of differential

diagnostic importance, weakness, emaciation and a decrease in the number of red blood corpuscles predominate. Both diseases may, however, sometimes occur simultaneously in the same patient.

Treatment. Besides the sanitary and dietetic measures noted elsewhere (see page 666), which yield very favorable results, Reinhardt makes subcutaneous injections of strangles serum (200 cc. followed by one to several repeated doses of 100 to 150 cc.). By these measures he decreased the mortality from 27.4% to 19.25%. Reinhardt, as also Brucker and Boesch, obtained much better results by intravenous injections of corrosive sublimate (4 to 6 injections of 80 cc. of a 1:1000 solution at body temperature at 24-hour intervals). In the author's cases this treatment was found to be unreliable. More recently intravenous injections of incarbon Merck (suspension of animal charcoal in sealed ampules of 40 cc.) have been recommended, in daily doses of 80 to 120 cc. for several days, besides intratracheal sprays of disinfectants in oily solution (see page 611). Poret had good results from intravenous injections of 2.5% phenol solutions (at first 10 cc. twice daily, then increasing by 10 cc. up to 150 cc.). Based upon his microscopic observations, Ruether recommends the use of trypanocidal substances (trypan-blue, trypan-rot, antimony, etc.), Friedheim gave 10% creosote vasoliment intratracheally (30 gm. daily for several days).

Prophylactic measures indicated are isolation of the well animals, general disinfection of the barns and utensils, use of separate watering buckets, and prevention of chilling and overworking of the animals. In case of a threatening epizootic, the body temperature of all horses should be taken daily.

Atelectasis of the Lungs. This occurs very frequently among domestic animals. Simader, who studied this affection in the Leipzig slaughtering house, found 15% of the slaughtered young pigs affected; 50% of sucking pigs; 7% of young goats; sucking calves, 13%; young sheep, 16%. Atelectasis may be primary or congenital, or secondary and acquired. Congenital atelectasis is due to the fact that portions of the lung do not expand after birth and remain airless during extra-uterine life. Acquired atelectasis occurs if some bronchi become impervious (obstruction atelectasis) or if portions of the lung become pressed upon (compression atelectasis). In both cases the air is absorbed within a few hours and the portions affected become void of air. As a third form we may mention marantic atelectasis which also is due to a gradual absorption of the air out of the alveoli. Simader believes that atelectasis in food animals is a congenital affection.

The **cause** of congenital and marantic atelectasis is usually an atrophic condition of the respiratory muscles due to fatty degeneration, lack of development, or due to various diseases of infancy (dysentery, polyarthritis, rachitis, rheumatism); not uncommonly atelectasis can be referred to insufficient physical exercise if the animals are kept continually in the barn. Obstruction atelectasis usually occurs during bronchial catarrh, more rarely in new-born animals, in consequence of

the obstruction of bronchioles by epithelial plugs, by mucus or by aspirated meconium. Compression atelectasis is usually caused by an accumulation of fluids or air in the pleural cavity, more rarely by voluminous tumors of the lungs or pleura.

Anatomical Changes. The seat of atelectasis is usually in the upper portions especially the anterior lobes of the lungs. The atelectatic foci appear browned, sunken in under the surface of the lungs and wedge-shaped if they are due to a bronchial obstruction. Congenital atelectasis is characterized by its typical location in the apices, by a normal appearance of all bronchi, or at least of those in the neighborhood of the atelectatic focus, and also by the fact that the pleural covering does not appear folded. If a portion of lung has not been expanded properly, hyperemia occurs and soon a serous infiltration of the atelectatic focus (splenization) develops; the epithelia then undergo fatty degeneration and become desquamated; mucoid material collects in the bronchioles and leucocytes wander into the interstitial connective tissue (desquamative catarrh). The tissue elements which have undergone fatty degeneration are absorbed and this is followed by marked atrophy which may progress to a degree that the two opposite leaves of the pleura finally touch (induratio).

There exist no observations concerning the clinical **symptoms.** It is, however, probable that in extensive atelectasis disturbances of respiration and physical changes in the thoracic sounds may be found similar to those encountered in catarrhal pneumonia. Congenital pulmonary atelectasis must always be suspected when animals are apparently stillborn. Atelectatic foci of lesser extent do not cause any symptoms at all.

The **significance** of the lesion depends upon the underlying cause. Atelectasis is of a certain importance because it might be confounded with contagious bronchopneumonia of young animals and an inflammatory process may be established secondarily in the atelectatic focus (usually bronchopneumonia, exceptionally croupous pneumonia), due to the action of non-specific microorganisms. Specific microorganisms may sometimes invade an atelectatic focus. (Hogs develop the pneumonic type of hog cholera in this way.)

The **treatment** of acquired atelectasis depends upon the cause. In congenital atelectasis one must attempt to stimulate respiration by massage of the heart (compression of the cardiac regions from both sides about fifteen to twenty times per minute), by artificial respiration (movements of the bent front leg upward and downward and then backward toward the region of the flank), the removal of mucus from the buccal cavity, rhythmical pulling out of the tongue, friction of the skin, instillations of vinegar into the nasal cavity, titillation of the nasal mucosa with a straw (Moussu). Hygienic measures, particularly proper breathing and proper care, are important from the standpoint of prophylaxis.

7. Enzootic Pneumonia of Young Animals.

(*Septic pneumonia of calves, lambs, kids, pigs, foals; enzootic bronchopneumonia of young animals, pleuropneumonia septica* [POELS], *pneumo-enteritis septica* [GALTIER]; *Ferkelsterbe, Ferkelhusten, Zementhusten, chronische Schweineseuche* [German]; *Bronchopneumonie infectieuse des veaux, des agneaux, des chevreaux* [French]; *pneumonite dei neonati* [Italian].)

Enzootic pneumonia of young animals is an infectious pneumonia, or pleuropneumonia, which occurs enzootically, and is usually catarrhal, not uncommonly catarrhal croupous, or purely croupous; it is caused by various bacteria, among them the bacillus bipolaris septicus and its varieties.

Though some of the forms of pneumonia here enumerated have already been described in Volume I. as special diseases, the collective consideration in one chapter of all contagious pneumonias occurring in young animals cannot be avoided, because only in this manner is it possible to do justice to the requirements of practice and also to consider those forms of contagious pneumonias which were not taken up in the other volume.

Historical. Reports on the occurrence of a contagious pneumonia in young animals are found since the middle of the last century; but they received more particular attention since 1890. In 1886 Poels described the disease in cattle in Holland and claimed as its cause a bacillus similar to the bacillus of swine plague. These findings have subsequently been confirmed by Jensen (1890), van den Maegdenbergh and Liénaux (1892). Perroncito, in 1885, described a pleuropneumonia of calves, occurring sometimes sporadically and at other times epizootically. He claimed as its cause an organism which he called micrococcus ambratus, a bacterium which is different from the bacillus bipolaris septicus. The exact etiologic position of the disease described by Perroncito has not yet been cleared up satisfactorily.

Aside from those named, the following have studied the etiology of contagious pneumonia in young animals: Semmer (1888); Bongratz (1892); Stroese & Heine (1898); Grips, Glage & Nieberle (1904); Schreiber (1906); Pfeifer (1906); Hartl & Reisinger (1907); Lignières (1907); Berger (1907); J. Müller (1907); Jensen (1911); and Kovarzik (1912).

Occurrence. Enzootic pneumonia of young animals occurs in all countries in which young animals are bred in large numbers and it has been observed particularly in Holland, Germany, Denmark, France, Belgium, England, Italy, Switzerland, Russia, Hungary and America. Most commonly affected are calves, lambs, young pigs, less commonly kids, and rarely foals. The animals are, as a rule, affected during the first weeks of their lives, but even up to the sixth month and later.

The disease has become of greater economic significance

since it recurs annually again and again in spring and becomes more extensive as a barn enzootic, causing numerous deaths and retarding the development of those animals which survive.

Etiology. From the standpoint of causation, the disease may be divided into two main groups. The decidedly infectious forms are directly produced by microorganisms that are, or have become, pathogenic. Among these, mention should first be made of the variant species of Bacillus bipolaris septicus, such as vitulisepticus, ovisepticus, suisepticus, and equisepticus. The Pneumobacillus septicus described by Galtier is probably identical with these microorganisms.

Pathogenicity of the Bipolar Bacilli. According to the experiments of Poels, the injection of Bacillus vitulisepticus into the lungs or serous body cavities killed rabbits in from 15 to 35 hours and calves in from 20 to 66 hours. Following intrapulmonary infection, a serofibrinous pleuritis sometimes accompanied by lobar pneumonia developed. Rabbits, guinea pigs and mice, when inoculated subcutaneously or when fed with cultures, die from septicemia. Artificially infected hogs sicken with symptoms of swine plague, while in other animals and sometimes in guinea pigs (Jensen) only abscess formation takes place at the point of inoculation. Dogs resist artificial infection. Positive results were also obtained in the experiments of Galtier with cultures of Pneumobacillus septicus in calves, lambs, kids and pigs. Semmer, on the other hand, did not succeed in transmitting the disease of calves to other healthy calves by nasal, intratracheal or intrapulmonary injection of tissue juices from the lungs. In Grave's case the undescribed bacteria *cultivated from the diseased lungs* were *without effect upon* hogs and small laboratory animals. The same observations were made by Hartl and Reisinger following the injection of a pleomorphic bacterium resembling Bacillus bipolaris. Upon subcutaneous inoculation of cultures of this organism, otherwise susceptible domestic animals did not become sick, but a pneumonia was sometimes produced in rabbits (Stoehr), as will sometimes also happen when such animals are inoculated with Bacillus **suisepticus (Smith, Kitt) and Bacillus bovisepticus (Marek).**

Several investigators found other bacteria, aside from the bacillus bipolaris as the cause of enzootic pneumonia in young animals. Poels, J. Müller and Schreiber observed an enzootic pneumonia in calves and young pigs, caused by a bacterium of the coli group. Poels & Berger described an enzootic pneumonia in calves and young pigs, caused by streptococcus pyogenes. Bacillus pyogenes may likewise occasionally produce enzootic bronchitis and bronchopneumonia in young pigs and calves (Grips, Glage & Nieberle, Olt, Poels, Berger). The bacillus bronchiolitidis vituli described by Kitt is probably identical with the

bacillus pyogenes. Poels also ascribes the faculty of producing enzootic pneumonia in young pigs to a staphylococcus pyogenes and to a kind of streptothrix, which he has not described more fully. Finally Berger observed an enzootic pneumonia in calves which was produced by bacillus pyocyaneus.

The causal relation between enzootic pneumonia and varieties of the bacillus bipolaris septicus is now generally considered as being well established. Still, many authors do not believe that the other microorganisms mentioned are at all etiologic factors in the production of enzootic pneumonia and hold that they are present only secondarily, having invaded an already diseased lung. This view appears justified in those cases in which bacteriologic examinations were made only in an advanced stage. However, other bacteria than the bacillus bipolaris have been found in the absence of the latter in very recent cases (Poels, Berger, J. Müller, Kitt, Grips, Glage & Nieberle, Olt) and pneumonia has been produced with them in experimental animals by subcutaneous and by intratracheal injection, as for instance by J. Müller with a bacillus of the coli group and by Junack with a staphylococcus or a streptothrix organism.

Several kinds of bacteria appear to be effective in some cases. Frequently, however, the presence of bacteria different from the bacillus bipolaris simply represents a secondary infection, particularly when the disease has run a longer course. This furnishes the basis for further tissue changes which may so modify the original pathologic picture that it appears as if we were dealing with a new disease (Holth). A secondary infection of the lungs may occur also in other enzootic diseases (pyosepticemia, dysentery of sucklings, white scours, strangles) and then give rise to numerous cases of bronchopneumonia (secondary bronchopneumonia of sucklings).

The Etiological Relation Between Enzootic Pneumonia of Young Animals of Various Species. Some incontestable clinical observations furnish the proof that enzootic pneumonia may occasionally be transferred from the young of one species to that of another. Lambs were infected from hogs suffering from swine plague (Keleti, Pr. Vb.). Calves were infected from calves, kids or foals suffering from septic pneumonia (Perroncito, Sauer, Pr. Vb., 1905, ii, 17, Bass, Pfeil); Rühm & Schreiber likewise consider the spreading of swine plague to calves, as probable or of calf-pneumonia to young pigs. In connection with the positive inoculation experiments mentioned, and the biologic properties of bacillus bipolaris, these observations permit the conclusion that the different forms of pneumonia caused by it in calves, pigs, lambs and kids are intimately related or are produced by varieties of the same bacterium. The bacillus bipolaris appears to have adapted itself, in the majority of cases, to a certain species, so that only the young of the same species are susceptible to contagion. If, however, its virulency becomes increased, or if certain environmental conditions become favorable to microorganisms, it may spread to young animals of another species, and exceptionally to adult animals of this kind. (Pr. Vb., 1905, ii, 17.) Young animals may also be infected from adults, as occurs in hem-

orrhagic septicemia of cattle, in swine plague and in hemorrhagic septicemia of sheep.

The same general rule might apply to pneumonia of the young caused by other microorganisms, but observations proving this are still lacking.

Natural infection occurs by sucking an unclean udder, through contact with straw, by licking the walls, through ingestion of infected food, through inhalation of droplets of secretion, which are coughed up by sick animals, through the stump of the umbilical cord, through abrasions of the skin. Sick animals confined to a stable will spread the infection by expectorated bronchial secretion, by intestinal discharges which may contaminate the floor, the straw, the feed remnants, the crib, and which may remain virulent upon the infected objects for a long time. *The disease has, therefore, a tendency to become stationary* in localities which have once become infected, unless prophylactic measures are carried out. The possibility of intrauterine infection, which was mentioned by Galtier, has received support by recent observations of Kovarzik. The importation of the disease into hitherto free herds is usually brought about by young or adult animals which have passed through the disease, yet have not fully recovered. The disease is not rarely spread to new localities by obviously sick animals or by the attendants. It is spread most easily by sick animals of the same species.

The apparently more frequent noninfectious forms of the disease develop under the determining influence of weakening conditions as the result of saprophytic organisms colonizing in the animal body or living in the environment. These organisms, after passing through the previously weakened animal body, may finally also become pathogenic spontaneously. These forms occur in young animals without necessarily being introduced from the outside, and weakening influences play a major rôle in their development, inasmuch as the micro-organisms which are absolutely necessary for the production of an inflammatory process in the lungs cannot exert their pathogenic action without the presence of such influences. Among such weakening influences there may be mentioned cold, confinement to cold, damp, draughty, poorly ventilated barns or other inclosures, and permanent stabling. Accordingly, the disease occurs more frequently during cold, wet, foggy seasons, and in cases where the animals have little or no opportunity to exercise in the open. Csonka and Szily observed the occurrence of an enzootic bronchopneumonia in young colts after they had been treated with a liniment made of crude oil and lime water. Other predisposing causes are digestive disturbances, especially at weaning time; *irrational composition of the feed*, especially with respect to the ratio between protein, fat and carbohydrates, as well as the content of salts of lime and phosphates and alkalies in general; also diarrhea and rachitis, which, together with confine-

ment to cold quarters with cement floors, are, in some regions, the most frequent predisposing causes in young pigs. Too great refinement in breeding, as well as congenital atelectasis of the lungs also constitutes a predisposition to the disease.

Pathogenesis. Some of the pathogenic bacteria mentioned above, especially bacillus bipolaris, multiply, in certain cases, very rapidly in the blood of the infected animals, and thus produce septicemia. If the latter does not lead to death in a short time some of the bacteria circulating in the blood become colonized in the bronchi and the pulmonary tissues and bronchitis and pneumonia develop. In the meantime, the bacteria may have disappeared entirely from the circulating blood and remain only in the organs where pathologic changes are established. Other bacteria, and also the bacillus bipolaris, may produce pneumonia without a preceding septicemia by gaining access to the lungs either with the inhaled air or through the blood-current. The localization of bacteria from the circulating blood or their dissemination with the lymphatic current may cause inflammation of the serous membranes of the thorax, particularly if the bacteria possess a higher degree of virulence.

Since diseased pulmonary tissue forms a good nutritive soil for various bacteria, saprophytic microorganisms living in the bronchi of otherwise healthy animals or in their neighborhood may multiply rapidly (secondary infection) and produce further changes.

Anatomical Changes. These vary according to the several microorganisms and their variable degree of virulence, also according to the power of resistance of the affected animals and to the duration of the disease.

Aside from those cases which run their course as a pure septicemia and which are not here considered, in its **acute form** the disease not uncommonly presents the picture of a septic pleuropneumonia. A serofibrinous exudate is found in one or in both pleural cavities, the pleura being covered to an extent of several millimeters with loose, juicy, fibrin membranes, which are lusterless and studded with punctiform hemorrhages. The lung is uniformly dense, void of air, friable in its anterior lower portion; frequently also to a greater extent, a reddish-gray cloudy fluid may be scraped off the dark red-brown or grayish-brown, finely granular cut surface. The interstitial connective tissue appears uniformly serously infiltrated and forms yellowish, gelatinous, reticulated stripes of varying width on a red-brown or more grayish-brown background (marbled). Sometimes one sees lymph vessels with coagulated lymph and a fibrinous exudate in the wider stripes (Liénaux). Of other changes occasionally met with may be mentioned gelatinous infiltration in the neighborhood of the larynx and the pharynx, hemorrhages into the tissue of the pericardium and peritoneum,

fibrinous pericarditis, acute swelling of the lymph glands, acute gastro-intestinal catarrh, sometimes ulcers in the abomasum (Beresow), cloudy swelling of parenchymatous organs, also, *according to Galtier, hemorrhages into, and fatty degeneration of*, the muscles of the rump, the extremities and of the heart. The anatomical picture in sheep and goats is sometimes similar to that of *septic pleuropneumonia*; in young pigs, however, to that of typical swine plague (see Vol. I).

Very frequent findings in acute cases are bronchitis or bronchopneumonia with or without a simultaneous pleuritis or pericarditis. Individual lobules or occasionally whole lobes (especially the anterior and median lobes and the anterior angle of the principal lobes) appear brown, purple or grayish-red, fleshlike, dense, non-crepitant when cut into. The interlobular connective tissue appears occasionally somewhat swollen; the bronchi contain mucoid, gelatinous yellowish masses of secretion. The bronchial and mediastinal glands are markedly swollen.

The **chronic form** presents a flabby pneumonic consolidation. Occasionally one finds in the affected pulmonary tissue purulent or caseous foci from pea to walnut size, in other cases dry, grayish yellow necrotic foci, sometimes also cavities as large as a fist. The pleuræ present the picture of a chronic fibrous adhesive inflammation. The bronchial and mediastinal lymph glands often show considerable chronic swelling and exceptionally contain dry foci up to the size of a lentil.

Pneumonia assumes the form of bronchopneumonia suppurativa if caused by bacillus pyogenes (Olt, Holth) or bacteria of the coli group (J. Müller). It is then characterized by a light yellow suppurative condition of the small bronchi, in the middle of the hepatized or only atelectatic lobules. A gradual purulent liquifaction of lobules, inspissation of pus and adhesive pleurisy radiating from the bronchioles takes place in the further course of the disease.

Kitt described a caseous bronchopneumonia in a young steer which was very similar to pulmonary tuberculosis, but differed from it in that there were no caseous foci in the connective tissue between the white yellowish caseous looking lobules; calcareous changes and cavity formation was likewise absent. The exudate in the bronchi contained exclusively the bacillus bronchiolitidis vituli (see page 685).

Symptoms. The duration of the period of incubation varies a good deal according to the type and virulence of the pathogenic bacteria, and it can be determined only in cases with an acute course.

According to Schreiber septic pneumonia occasionally appears on the second day of life; in other cases on the ninth to seventeenth day after infection. In contagious catarrhal pneumonia of young pigs Grave observed a period of incubation of ten to fourteen, rarely up to twenty-four days.

The clinical picture itself varies a good deal. One can distinguish, generally speaking, an acute and a chronic form of the disease.

The **acute form** is usually observed in very young animals

a few weeks old at the utmost; it is initiated by a decrease in the lively behavior and in appetite, and by a febrile elevation of temperature up to 41° to 42° C. The respiration is accelerated from the start and becomes more and more frequent, so that after a few days the number of respiratory movements may be 40-60 even 100 per minute. At the same time the respiration becomes forced, the animals spread their front legs, stretch their heads and neck; calves and foals also dilate their nostrils. Cough occurs soon after the onset of the disease; at first it comes on during exercise; later on also during rest and it becomes more frequent and more tormenting. Mucopurulent nasal discharge occurs in some cases.

Percussion and auscultation of the chest reveal the same changes as are usually found in bronchopneumonia (see page 681). In some cases there are found in addition the signs of acute fibrinous or serous pleurisy or pericarditis. Exceptionally the disease may present itself exclusively under the clinical picture of acute pleurisy or pericarditis (Immelmann).

The physical weakness increases rapidly during the course of the disease; the animals become unable to stand up and they succumb between the second, fourth, sixth day of the disease; often profuse and fetid diarrhea has set in. If, as occurs rarely, the animal remains alive the symptoms of the chronic form of the disease are developed.

The **chronic form** is seen in somewhat older animals or in somewhat milder outbreaks; cough being the first obvious symptom of this form of the disease. It is at first infrequent and comes on only on rising or lying down or during exercise. Later on it becomes more frequent and weaker. In the meantime, the respiration becomes more frequent and more or less forced. Râles and whizzing sounds, also purring are heard over the chest and in addition often the changes on percussion and auscultation as in the acute form. Fever is often present; but only of moderate degree. The general condition and the appetite may remain undisturbed, particularly in hogs; however, the appetite is, as a rule, diminished and the sensorium more or less depressed.

Gradual emaciation becomes manifest in the further course. In this manner a chronic marasmus is established, which may terminate fatally in three to six weeks or only after one to two months. In the milder cases the condition gradually improves, the cough becomes less frequent, the nutrition better, and all symptoms entirely disappear, often however, only after several months.

In some cases, apparently in those due primarily or secondarily to the bacillus pyogenes, itching and scabby eczemata, appear on the neck along the back, on the root of the tail, also swellings and abscesses in various parts of the body, which are, according to Schimmelpfennig, of unfavorable prognostic significance.

Diagnosis. The exclusive affection of young animals and the enzootic occurrence of the disease are generally sufficient

to lead to a correct diagnosis, and there is frequently besides the additional chance to make a post-mortem examination on one of the earliest fatal cases. The exclusive affection of calves, lambs or kids distinguishes the disease from pleuropneumonia in cattle and from hemorrhagic septicemia of sheep and goats. The same is true of the differential diagnosis between contagious pneumonia of young pigs and typical swine plague which is caused by bacillus suisepticus alone, while in hog cholera the clinical and anatomic pictures are usually characteristic. A favorable course also speaks against swine plague. In doubtful cases the epizootical factors have to be considered. Contagious pleuropneumonia can usually be easily distinguished from pneumonia of calves according to the principles laid down in Volume I. An enzootic occurrence permits the exclusion of sporadic bronchopneumonia. Lungworm disease is usually seen in somewhat older animals after pasturing. The detection of worms, their embryos and ova in the coughed up sputum, or on post-mortem examination in the bronchi, establishes the diagnosis beyond doubt. Atelectasis of the lung must also be considered in the anatomical diagnosis (see page 683). The fact that calves and lambs hold their bodies stiff might lead to a confusion with tetanus; a more exact examination will, however, exclude such an error.

The detection of the causative microorganism should be attempted in all cases by post-mortem examination and by bacteriologic tests of recent cases.

Prognosis. It is usually quite unfavorable; however, considerable variations occur in various enzootics as to the morbidity and mortality, according to age, species and environmental conditions of the animals. Most of the animals which are affected during the first weeks of life perish and even those that remain alive are stunted in their development, and their raising does not pay. A complication of dysentery is particularly unfavorable. The disease very often terminates favorably in young pigs over one month old (Greve, authors' own observation), and also not uncommonly in other animals; the recovery may, however, be apparent only and extensive indurations may persist in the lungs.

Treatment. Medicinal treatment is not at all promising and its carrying out meets with great difficulties. Dietetic and hygienic regulations may, on the contrary, influence the course of the disease favorably (see page 666). Animals which are not very seriously ill often recover completely in a relatively short time, if they are kept permanently in the open air, which is possible even in winter, according to Kovarzik, when the weather is favorable. Less severe cases were cured also by prolonging the period of suckling, by generous feeding of the weaned animals, especially with milk; shearing sometimes has a good influence on the nutrition of less severe cases.

Serum treatment may be of value in those cases of slight or moderate severity in which the origin can be determined by investigating the pathological changes, and especially through the bacteriological examination of recent cases. Curative and prophylactic sera should be given subcutaneously, singly or in combination accordingly as the infection is simple or mixed; the injections are repeated as needed, once or several times a week, double the dose required for prophylaxis being selected. In general it is best not to count upon definite curative results from serum treatment, unless the hygienic conditions are improved as well; this treatment is therefore of subordinate importance.

Protein therapy in the form of the subcutaneous injection of serum foreign to the species (normal horse serum and anti-anthrax serum from horses), or the serum of animals that have recovered from hemorrhagic septicemia (20 cc.), was repeatedly used with good results by Kovárzik. Csonka and Szily used typhoid vaccine.

Prophylaxis. Prophylactic measures carried out energetically permit the successful struggle against enzootic pneumonia of young animals; hence our main efforts must be spent in this direction. To prevent the importation of the disease, it is advisable not to introduce too young animals for breeding purposes and to subject newly bought stock to an isolation of two weeks and then to keep them together with other young animals only provided they are healthy and have not shown any suspicious symptoms (cough!). The animals should be bred and kept in as natural a manner as possible, in order to increase their resistance. In this respect it is important to keep the stables dry, to clean and ventilate them daily; the floors should be flushed with whitewash after cleaning, and then covered freely with bedding; the floors must be kept warm for sows with young and, in case of need (cement or stone floors), wooden floors may have to be laid. The young animals are to be kept in the open air, if possible in pasture, if the weather is favorable. The mother animals and the weaned young must receive suitable food and, particularly in hogs, the calcium-requirements need consideration. The weaning may be postponed; excessive refinement in the breeding and inbreeding are to be avoided.

The occurrence of this and other infectious diseases has frequently been prevented in calves by tying them separately near their mothers. The same purpose is served by the use of a specially constructed wooden auxiliary barn with a separate box stall for each calf, as recommended by Titze. Such quarters may be built in the cowshed and so constructed that the calves, when 8 days old, may pass at will directly into a large box stall which is separated by a wooden wall from the cow shed proper. Plenty of exercise in the open (if possible with pasturage) is especially important. This can easily be provided by building

a shed against the south wall of the cow barn with only a communicating door. The calves can then be allowed to pass at will from such a stall into an inclosure to the south. In the management of calves and colts at pasture in the winter, the method recommended by Schneider and Hink, in which the young animals are accustomed to the rough weather from the very beginning, deserves special consideration. All animals born between April and October are immediately placed into an inclosure with shade trees and a roof for shelter, while calves and colts born in the winter are placed in cold box stalls and daily driven out into the open. This plan offers the veterinarian a better prospect of success in protecting the young stock against the maladies in question than does the use of protective vaccines.

If, in spite of precautions, the disease has made its appearance, healthy animals should be separated from the sick and suspicious ones. The removal of the healthy animals and their mothers to another stable or another farm, etc., where there are no young ones is very desirable. Those animals which are to be transferred should be cleaned, especially on their extremities. The isolated animals should have separate attendants. If the disease has made its appearance to a limited extent, it is best to slaughter the sick and suspicious animals, otherwise they may be kept alive and isolated until they either recover or until their slaughter becomes necessary. Recovered animals should only be used for breeding purposes with every possible precaution, since they may become instrumental in spreading the disease.

Stables, pens and runs must be disinfected thoroughly after the removal and burning of loose woodwork. Manure must be made innocuous by the use of chloride of lime. Stables for breeding animals, which have been visited repeatedly by the disease, must be disinfected before the young ones are born (the procedure after parturition has been indicated in Vol. I).

These measures usually make it possible to stop the spread of the disease and to prevent its occurrence.

Protective Vaccination is of limited importance, at present, as compared with the hygienic and prophylactic measures, although it is impossible to decide definitely upon the value of the method, the many unfavorable results being probably due, in part, to a now critical employment of the vaccines without reference to the causal virus. The administration of protective vaccination is to be considered particularly in stock where the other prophylactic measures cannot be carried through or where their beneficial influence is to be supplemented. In order to insure satisfactory results, the nature of the causal virus must be determined for each enzootic by bacteriological examination of recent cases, best in a well appointed laboratory

and with reference to the pathological changes, and the vaccine must be determined upon accordingly. Since different strains of any bacterium may have different characteristics, it seems that only polyvalent protective vaccines are suitable for practical use, and these seem to act reliably if the proper variety is used in a given case. The sera are employed singly or combined accordingly as the infection is simple or mixed. Various protective sera are employed to produce a passive immunity lasting two to three weeks; a more prolonged active immunity may be caused by means of bacillary extracts (vaccines) alone, or combined with immune serum (simultaneous vaccination); in some cases the passively immunized animals may be left in the infected stable. The immune sera are recommended generally in enzootics with an acute course, the bacillary extracts in simultaneous vaccination are preferable in slow protracted enzootics. The active immunization of the fetus through that of the mother by means of bacillary extracts has not been found practicable. In infected herds, protective vaccination should be practiced immediately after littering, if possible before the first suckling. It is to be repeated in two or three weeks once or twice, or two weeks before weaning, especially if only immune sera are administered.

The following specific remedies are to be considered (see the resp. chapters in Vol. I for particulars):

Septic pneumonia (caused by Bac. bipolaris); Polyvalent serum of pneumonia of calves (for calves 10 cc., for lambs 5 cc., for colts, 20 cc.); polyvalent, germ-free bacillary extract of septic pneumonia (for simultaneous vaccination with the above amounts of protective serum, for calves 5 cc., for lambs, 2 cc., for colts, 10 cc.; during the 5th week of life it is given alone in amounts of 8, 3 and 15 cc.); Prophylactic serum for pneumonia (for calves 10 cc., for lambs, 5 cc., for colts, 15-20 cc.); curative serum for pneumonia (same doses); curative lymph for septic pneumonia (same doses); polyvalent serum for swine plague (5 cc. to young pigs); polyvalent, germ-free bacillary extract for swine plague (in doses of 2 cc. for simultaneous vaccination during the first days of life, with 4-5 cc. of prophylactic serum; 14 days before weaning a further dose of 3 cc.); curative lymph for swine p[illegible] (5-20 cc. to young pigs); Suptol (5 cc. to young pigs); Serum and vaccine for swine plague (simultaneous vaccination similarly as with the preceding preparation). Euman (a combination of prophylactic serum—B. suisepticus and B. pyogenes—and bacillary extract; newborn pigs are given 3-4 cc. subcutaneously; if necessary the injection may be repeated after 2 to 3 weeks; in any case 5 cc. are to be injected before weaning. The therapeutic dose is 1-2 cc. for 5 kgm. of body-weight, not less being given than 5cc.). Kovarzik saw favorable curative results in several cases of septic pneumonia of calves after the subcutaneous injection of serum of animals which had recovered from hemorrhagic septicemia (20 cc.), also of serum from healthy horses and of anthrax-immune serum.

Paracoli-pneumonia; Polyvalent paracolon bacillus serum (to calves 20 cc., to lambs 10 cc., to colts 30 cc.).

Colon bacillus pneumonia. Polyvalent colon bacillus immune serum; (dysentery, antidysentery serum) in similar doses as the preceding serum.

Pyogenes-pneumonia; Pyogenes-serum in similar doses as above; Enman (see above).

Streptococcus-pneumonia; Polyvalent streptococcus serum (glanders serum) for colts in doses of 10 cc., 4 to 5 days later, bacillary extract of glanders (5 cc.).

8. Mycotic Pneumonia. Pneumonomycosis.

(*Pneumonomycosis aspergillina.*)

Pneumoconiosis is a bronchopneumonia produced by moulds, which is followed, in birds, by mycosis of the air sac.

Etiology. Moulds in the air passages are found most frequently in places where the temperature is somewhat lower and where fungi can remain permanently in contact with the air. They also form larger colonies in the presence of more abundant masses of secretion or decomposition products. One finds colonies of moulds in bronchiectasias, pulmonary cavities, exceptionally in bronchial catarrh. In all of these cases, however, *moulds live in the interior of the air passages only as* saprophytes without attacking the living tissues and without producing pathologic changes in them (secondary mycosis).

Only rarely can disease of the air passages be referred directly to a pathogenic effect of moulds (primary or true mycosis). This appears to be the case after the inhalation of very great amounts of moulds. Moulds may display a pathogenic effect preferably in weakened individuals and in the presence of a catarrhal affection of the lungs. Schultz produced the disease artificially in birds by inhalation. According to Fölger, mould spores may also get into the lungs by *embolic transport.*

Most common of the pathogenic moulds are species of aspergillus, especially aspergillus fumigatus and aspergillus nigrescens. The former still grows well at a temperature of 37°-40° C.; the latter at 37° C.; hence aspergillus fumigatus *is considerably more dangerous (Schultz, Lucet). Aspergillus* glaucus does not grow at body temperature and only forms larger colonies perhaps in the larger air passages, but it cannot *penetrate into the tissues. Some species of mucor grow* at 40° C. and also in the interior of animal tissues.

The **species of aspergillus** form a colorless mycelium, from which arise straight, non-branching hyphæ; these end free in a globular swelling, the columella, which carries numerous radially arranged sterigmæ, from which a single row of conidia *is formed by constriction, which, in combination with the sterigmæ and the columella* form the fruit-headlet (Fig. 19). The following species are pathogenic:

Aspergillus fumigatus forms at the beginning bluish-green, later on ashy-gray colonies. The diameter of the semispherical or club-shaped columella is 8 to 20 μ; of the conidia 2 to 3 μ. The sterigmæ rise more or less upward.

Aspergillus nigrescens (asp. niger.) columella spherical and carries pure radially arranged sterigmæ, with blackish conidia. The mycelium therefore looks chocolate colored.

Aspergillus glaucus; the hypha ½ to 1 mm. long possesses a club-shaped columella with radially arranged sterigmæ. The segmented conidia are grayish-green, the mycelium is of the same color (Fig. 26).

Characteristic of **Penicillia** (brush-moulds) is an umbilliferous arrangement of the conidia bearers, which terminate in bottle-shaped cells, each one of which carries a chain of conidia (Fig. 27). Pathogenic penicillia have so far not been found in domestic animals.

The *fruit bearer in* **Mucorinae** *terminates in a spherical brown or black*

sporangium containing the spores. After rupture of the smooth membrane these escape into the outside world (Fig. 131). Mucor racemosus and mucor conoideus have heretofore been found in the air passages.

Mortierella parasitica was found, by Neumann, in the trachea of a cat dead of suffocation. It differs from mucor by the absence of the columella. San Felice found Cryptococcus granulomatogenes, belonging to the saccharomycetes, in cheesy foci in the lungs of a hog.

Natural infection takes place on living in musty, moist-warm, ill-ventilated spaces and in consequence of the ingestion of mouldy feed. Animals which get but little into the open air and which are feeble or weak appear to be predisposed. The disease is, therefore, most common among domestic fowls, since they are comparatively frequently kept in mouldy, moist, poorly ventilated places. Primarily affected are pigeons, chickens, ducks, geese, sometimes also other birds, and even cage birds (Johne saw pneumonomycosis in nineteen flamingoes of a zoological garden; other authors have seen the disease in parrots). Mycotic pneumonia has more rarely been seen in mammals; Schultz, Rivolta, Martin, Bollinger, Lucet, Peck saw the disease in horses; Röckl, Piana, Bournay, Ravenel, in cows; Hartenstein, in calves (in eleven calves in Hungary); Hellews, in sheep in enzootic extent; Mazzantini, in a lamb; Serrurier & Rousseau, in a stag; Rivolta, in a dog, and Waldmann in a hog.

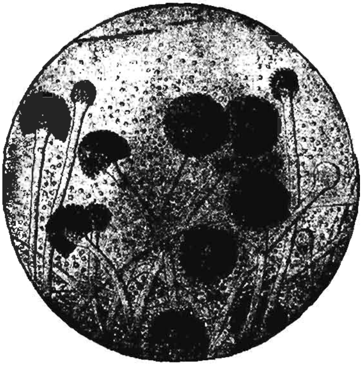

Fig. 129. Aspergillus glaucus.

Anatomical Changes. Dirty yellow or greenish, mouldy looking, dry deposits are formed on the mucosa of the bronchi and on the internal surface of the air cells in birds; under these deposits the tissues are sometimes ulcerated and they may completely obstruct the bronchial lumen. In the pulmonary tissue catarrhal foci are sometimes found; sometimes disintegrated tissue composed of purulent caseous or more grumous nodules and surrounded by a connective tissue capsule or by an infiltrated, occasionally hemorrhagic, focus, which is at other times imbedded in a hepatized tissue (Martin). In birds, grayish-yellow fibropurulent, disc-like or flattened masses of exudate may be found between the surface of

the lungs and the wall of the chest; or, again, the air sacs may be converted into yellowish, cheesy, bladder-like structures, from the size of a pigeon's egg to that of an apple, with a yellowish to green, sometimes soft, deposit on the surface. Lange, on the other hand, found in geese, chickens and pigeons homogeneous tuberous foci up to the size of a pea within the lung tissue, which was otherwise apparently healthy, while the usual changes in the respiratory passages were absent. Similar whitish, homogeneous, sharply defined granulomata were observed by Darmagnac and Barlette following the entrance of Saccharomyces, especially in the lungs and liver, but also in the peritoneum, intestine, kidneys and pericardium. In ostriches Jowett found only the lungs affected. They contained numerous small, yellowish-white, pearl-like nodules and showed hepatization; hence they presented the picture of an acute, miliary pseudotuberculosis. Paci found on the pleural surface of the lung in cattle a thin, greenish, smeary deposit. In a rabbit Schoeppler found changes similar to those observed in tuberculous cheesy pneumonia in cattle. On microscopic examination the deposits and the foci show the moulds. This differentiates definitely mycotic foci from caseous tuberculosis foci or glanders nodules, to which they may be very similar. Hepatization of neighboring nodules and of the interstitial connective tissue may in cattle simulate the picture of pulmonary tuberculosis (Röckl). In horses the changes may be similar to pulmonary gangrene (Pech).

Fig. 130. Penicillium glaucum.

Symptoms. In birds the respiration becomes rattling, gasping and more and more difficult. The appetite is poor, but the thirst is much increased. The animals do not like to move; their wings droop and they sit long in one place with ruffled feathers and somnolent. The buccal mucosa sometimes shows yellowish or greenish deposits (Potain). The emaciation progresses more and more; diarrhea comes on and the completely exhausted animals die after a period of four to eight weeks' duration.

Symptoms pointing to a chronic pulmonary disease are observed in **mammals** and may exist for weeks and months; the animal suffers from increasing respiratory difficulties, they become emaciated to skeletons and completely exhausted. More rarely the disease takes a rapid course and then leads to more acute symptoms, eventually with a hemorrhagic discharge from the nose and to hematuria (Thary & Lucet).

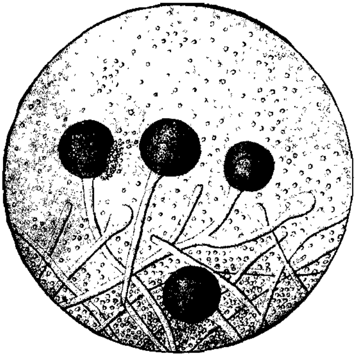

Fig. 131. Mucor mucedo.

According to Omlin, a mycosis of the skin due to Aspergillus is observed in horses, in the form of gradually enlarging tumors on the head and especially on the lips, exceptionally also on the eyelids, and which may extend to the adjoining mucous membranes. Such tumors appear as tough, almost painless indurations with a granular or knotty surface, resulting in gradual enlargement of the involved parts. Sometimes circumscribed abscesses are formed which lead to the formation of small ulcers and scars. There may also be observed salivation, mechanical interference with prehension of feed, a seropurulent discharge from the eyes and slight photophobia. On cross-section of the tumors, yellowish necrotic foci the size of a millet seed are seen in the septated parts. In the mucopurulent content from one to several grayish-white to sulphur-yellow granules the size of a grain of sand can be detected. On the margin of the small abscess the mycelium may be detected with a microscope.

Treatment consists in extirpation of the diseased tissue. As a rule even partial removal leads to recovery.

Diagnosis. The disease can be diagnosticated with certainty only after the microscopic detection of the moulds in the secretions of the air passages, after the history and the development of the disease have led to a suspicion of pneumonomycosis. Microscopic findings are insufficient, because similar moulds may be found in the air passages, particularly of mammals, as harmless saprophytes.

Treatment and Prophylaxis. To prevent the further multiplication of the moulds, inhalations with disinfecting solutions, or intratracheal injections (see page 610) may be tried. Their success appears very doubtful, because they do not reach the moulds which are located in the alveoli. One might try the methodical administration of iodide of potash.

Darmagnac and Barlette repeatedly observed recovery from saccharomycosis after the administration of 10 gm. of the following mixture: Tincture of iodin 10 parts, water 60 parts, potassium iodide 2 parts. The enzootic was entirely checked when the healthy animals were given 5 gm. of the same mixture every other day.

The most important measures are prophylactic. The animal houses, especially those of fowls, must be kept clean and dry, and the feeding of mouldy material must be avoided. To be recommended are good ventilation, drying out and cleaning of the places, washing of the walls and wooden portions with hot water or a disinfecting solution.

9. Purulent Pneumonia. Pneumonia suppurativa.

(*Abscess of the lung. Pneumonia embolica. Abscessus pulmonum.*)

Etiology. A frequent cause of purulent pneumonia is the lodgment of infected emboli, especially during the course of septicopyemia of sucklings. Other sources of infected emboli may be: purulent metritis after parturition, abscesses of strangles, ulcerous endocarditis, purulent or gangrenous wounds in various places of the body (pressure gangrene, gangrenous inflammation of the feet), fistulæ formed after venesection or castration, etc. Foreign bodies, and with them pyogenic bacteria, more rarely get into the pulmonary tissue. Such foreign bodies may enter the lungs from the air passages after injury to the thoracic wall from the stomach.

Suppuration of pulmonary tissue most rarely follows croupous or catarrhal pneumonia (concerning catarrhal purulent pneumonia caused by the bacillus pyogenes in cattle and swine, see page 671).

In some forms of catarrhal pneumonia or independently from it the bacillus pseudotuberculosis of Preisz forms greenish yellow, soft caseous foci (see Vol. I), and more rarely the bacillus necrophorus causes in catarrhal or croupous pneumonia more dry and caseous foci. Schlegel found numerous grayish yellow to grayish-red nodules in a dog; they contained the streptothrix (Actinomyces canis).

Anatomical Changes. In acute cases cavities are found in the lungs filled with pus and tissue detritus; their internal surface is uneven and villons, the surrounding pulmonary tissue is tough and void of air in consequence of cellular infiltration. On the other hand, whole pulmonary lobes may be infiltrated showing on the cut surface grayish-yellow points of the size of a pin head. Older abscesses have their own

connective tissue walls and well established cases show inspissated pus or dry caseous masses in their interior. The pleura shows evidences of a seropurulent or purulent inflammation whenever the abscesses are situated superficially.

Symptoms. The symptoms of the disease vary very much from case to case. If suppuration has followed upon croupous or catarrhal pneumonia, the liquefaction of the exudate is retarded, an elevation of temperature again occurs and the grave symptoms of acute pneumonia again stand in the foreground of the clinical picture. Embolic purulent pneumonia develops rapidly and leads in a short time to severe symptoms.

The symptoms point to grave acute pneumonia with high fever (Fig. 132) and respiratory disturbances; later on the signs of a septic infection are present. Percussion either shows no deviation from the normal or there may be more or less dullness, or tympanitic sounds in circumscribed places; eventually also metallic sounds. One hears râles, sometimes also bronchial or amphoric breathing. The picture of pulmonary gangrene is frequently developed.

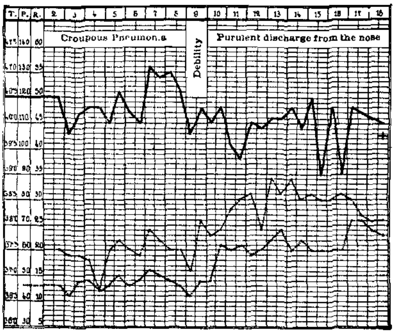

Fig. 132. Fever curve in purulent pneumonia in a horse.

The **diagnosis** can be established beyond doubt only if an abundant, purely purulent nasal discharge is present and if we find in it on microscopical examination elastic fibers (Fig. 133), fat crystals and possibly also necrotic pulmonary tissue. If an atypical pneumonia follows upon a purulent inflammation of internal organs, the existence of embolic purulent inflammation of the lung is to be taken into consideration particularly.

Course. The disease terminates, as a rule, fatally within a very short time; occasionally after only one to two weeks. The immediate cause of death is usually sepsis, also serofibrinous, purulent, sometimes gangrenous, pleurisy. Recovery is very rare, but it occurs occasionally. *Abscesses may become* encapsulated or they may break into a bronchus and finally heal, as was seen several times by Johne. It is quite difficult

to diagnosticate such slowly healing abscesses on the living animal.

Treatment is usually unsuccessful. In practice stimulating drugs may be employed, also disinfection of the air passages and pneumotomy in case of superficial abscesses.

10. Pulmonary Gangrene. Gangraena Pulmonum.

(*Gangrenous pneumonia, foreign body pneumonia, pneumonia gangrænosa.*)

By gangrene of the lungs is meant the putrid decomposition, by saprophytic bacteria, of pulmonary tissue which has previously become necrotic.

Etiology. Necrosis of pulmonary tissue is usually brought about by inflammatory processes, more rarely by injury to the lungs. Putrid bronchial catarrh frequently plays a rôle *and it usually develops after aspiration (aspiration pneumonia).* It occurs most frequently in horses, not as frequently in cattle, rarely in other domestic animals, and is then usually associated with disturbances of deglutition. As the cause of the latter there is usually found acute inflammation of the pharyngeal mucosa or of the adjoining organs. Disturbances of deglutition may be caused mechanically by abscesses in the pharyngeal wall or in the retropharyngeal lymph glands, by tumors, foreign bodies in the pharyngeal wall or in the air sac, paralysis of the pharynx, convulsions of the pharyngeal muscles (in tetanus or meningitis). Any disease associated with grave disturbances of consciousness may likewise cause aspiration. Aspiration occurring in paralysis of the pneumogastric nerve (as, for instance, in chronic lead poisoning—Thomassen) and so-called vagus pneumonia, following it, is dependent upon anesthesia of the larynx and the air passages, and upon paralysis of the muscles of the larynx, which makes the removal of the foreign bodies that might get into the larynx impossible. Nutritive disturbances of the pulmonary tissue may, according to Bettini, likewise take part in it. The sudden entrance of larger amounts of fluid into the pharynx may likewise give rise to aspiration as occurs in the breaking of a peri- or retropharyngeal abscess, in obstruction of the esophagus, during vomiting, in sudden falling down of cattle, in malignant foot-and-mouth disease; also in consequence of the protrusion of the contents of the paunch. In protracted labor a fetus sometimes aspirates amniotic fluid that is already contaminated. Foreign bodies, such as ears of grain, pieces of wood, hairs, are sometimes aspirated. In the pleural cavity of a dog, found dead from pulmonary gangrene, Mori once found six ascaris worms. He believed that they had been

aspirated in vomiting and had gotten into the lungs and from there into the pleural cavity.

Artificial feeding or the administration of liquid medicines may become dangerous in more serious disturbances of deglutition. Pneumonia caused by aspirated medicines may occur without the simultaneous existence of disturbances of deglutition (drenching pneumonia, pneumonia medicamentaria), if the medicines are administered awkwardly, if they are given at one time in too large doses, if the tongue is pulled out, or the nares are closed, if the head is held too high, if the drenching is continued in spite of cough or squealing (in hogs), if the fluid is poured into the nose, if the medicine cannot be swallowed easily, or if it produces attacks of cough.

Various, including putrefactive, bacteria, which in such cases have gained access with the aspirated substance into the bronchi, then multiply and produce first a severe inflammation in the bronchial wall, and the inflammatory process may be followed by necrosis and putrefaction. The inflammation spreads through the bronchial wall and also along it into the pulmonary parenchyma; here it produces catarrhal and hemorrhagic inflammation and soon also necrosis and putrefaction.

Of the various types of pneumonia, croupous pneumonia of horses and swine, preferably and comparatively frequently passes into pulmonary gangrene; catarrhal pneumonia leads less frequently to this process.

The contents of abscesses and caseous foci (tuberculosis, echinococcus, etc.) may become putrid; bacteria which have entered with the inhaled air are then responsible for this process. Infected emboli may carry the organisms (in Nielsen's case bacillus necrophorus) from other gangrenous or ulcerating organs into the lungs.

Injury to the lung may directly become the cause of pulmonary gangrene if putrefactive bacteria gain entrance at once or later on, or if portions of lung have been crushed. Injury to the lung may be brought about by external violence (wounds, fractured ribs) or, particularly in cattle, by foreign bodies which enter the lungs from the stomach or pericardial sac.

Foreign bodies do not always cause gangrene but sometimes only an interstitial pneumonia, as is demonstrated not so very rarely by findings on slaughtered animals. The foreign body may even be coughed up (Lisizin).

Anatomical Changes. Gangrenous foci, usually quite numerous, are found in the lungs, especially in their anterior and inferior portions. These foci appear dirty-brown red or dirty-yellowish brown; they consist, either at the periphery, or throughout their entire extent, of a mushy mass of very disagreeable, sweetish, foul smell. Liquefied or soft masses, which have a similar smell and are likewise discolored, are found also in the bronchi, the mucosa of which is dirty red or slate-gray

in color. In the neighborhood of the gangrenous foci and between them, the pulmonary tissue shows the picture of a catarrhal or croupous pneumonia. The internal surface of the cavities that are formed is ragged, eaten out, and is covered with a friable, smeary, stinking, mushy mass. In the rarer more chronic cases one finds in the neighborhood of the cavities a yellowish, or yellowish-gray, purulent, infiltrated zone or a cicatricial gray shell of connective tissue.

Foci situated near the pleura frequently lead to a purulent or putrid pleurisy, or pneumothorax develops after the focus breaks through. The internal organs present the signs usually found in grave general infection, such as parenchymatous and fatty degeneration, occasionally also hemorrhages and metastatic foci.

Symptoms. The *earliest symptom of pulmonary gangrene* is usually a peculiar, sweetish, foul, very disagreeable smell of

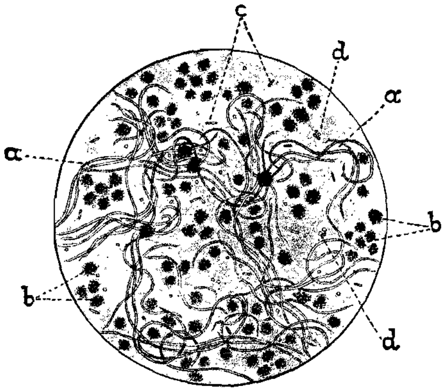

Fig. 133. Nasal discharge in pulmonary gangrene of the horse. *a*, elastic fibers from pulmonary tissue; *b*, pus corpuscles; *c*, bacilli; *d*, cocci.

the exhaled air. Stinking gases are developed in consequence of the putrefactive processes; these escape with the exhaled air and are at first noticeable only in the immediate neighborhood of the nose of the animals; later on they pervade the air of the neighborhood and can be smelled at once upon entering the place where the patient is kept. This smell may, however, be absent *in pulmonary gangrene, if the focus has remained closed* and has no communication with the outside world through the air passages.

As soon as gangrenous foci have broken into bronchi, there appears a dirty-grayish red, eventually brown-red or greenish tenacious nasal discharge, which becomes very abundant after coughing or after lowering of the head and which always disseminates the disgusting smell. Microscopically, the nasal discharge (Fig. 133) shows granular tissue, detritus, fat droplets, needle-shaped fat crystals, brown or black masses of pigment, sometimes pus corpuscles, red blood corpuscles, numerous varieties of bacteria, and elastic fibers, occasionally in alveolar arrangement. (Elastic fibers can easily be demonstrated after boiling the exudate in 10% solution of caustic potash, followed by centrifuging.)

Corresponding to the extent of the inflammatory process, the respiration is difficult and accelerated; this becomes particularly marked after a general septic infection of the organism has taken place.

The percussion sounds remain unchanged if central portions of the lung are affected exclusively. Frequently, however, we hear dullness, and occasionally also tympanitic sounds in the anterior and inferior pulmonary portions. The formation of cavities may sometimes be diagnosticated if elastic tympanic, or metallic sound, or cracked pot sounds are found in a circumscribed place of the thorax, associated with metallic râles and bronchial breathing; these correspond in pitch with the tympanitic percussion sound. Splashing and similar noises are also characteristic of the existence of pulmonary cavities.

Pulmonary gangrene is almost always accompanied by fever. Its intensity varies from case to case, but it usually reaches above 40° C. and shows a remittent type (see Fig. 134); an insignificant fever is often seen at first for several days in aspiration pneumonia. The pulse becomes accelerated, small, and soon thready. Chills and sweats come on from time to time. There is great prostration and dullness of the sensorium, the appetite is suppressed and profuse diarrhea not uncommonly comes on toward the end of the disease.

Pleurisy frequently develops during the course of the disease; pneumothorax only exceptionally. Pulmonary hemorrhage from the rupture of a blood vessel by coughing or by ulcerative processes of the vessel walls is rare.

Course. If pulmonary gangrene follows upon some form of pneumonia, it is preceded by the symptoms of this disease. If gangrene occurs in the course of croupous pneumonia, it usually occurs toward the terminal stage of hepatization; the non-appearance of improvement or resolution, or a new elevation of the temperature which had been falling, then point to an aggravation of the condition. It is also possible that the symptoms of pulmonary gangrene become manifest only after the beginning

of the stage of resolution; a putrid inflammation of parts situated more centrally may at first not become manifest and may only be suspected after the fetid odor of the exhaled air and of the nasal discharge have been observed. In all cases, however, after once being established, the gangrene progresses rapidly to a fatal termination, and the animals die between the second and fourth, or in any event before the end of the eighth day. Pulmonary gangrene arising after other diseases than croupous pneumonia, usually takes a similar course, except that sometimes weeks may elapse before a more considerable portion of the lung has become affected. The symptoms are then at first mild and only gradually increase in severity. A gangrenous pulmonary focus may, in exceptional cases, become encapsulated and recovery take place.

Fig. 134. Fever curve in gangrene of the lung in a horse.

Diagnosis. The only pathognomonic sign of pulmonary gangrene is the presence of shreds of pulmonary tissue or of elastic fibers in alveolar arrangement found in the nasal discharge or in the sputum expelled by coughing. Without the detection of these elements, the diagnosis depends upon the peculiar smell and upon the detection of signs pointing to cavity formation in the lung. In other cases, a diagnosis can be made only with some degree of probability; it becomes, however, fairly certain as soon as the peculiar disagreeable smell becomes manifest in the course of acute pneumonia, or if the symptoms *of pneumonia come on after a disease which points to the aspira-*

tion of particles of food, masses of secretion, etc. Signs of sepsis are also of importance and always accompany pulmonary gangrene of any extent.

The disease might be confounded most easily with putrid bronchial catarrh; if primary, this does not lead to any considerable elevation of temperature, or to pulmonary consolidation; it is, however, always present as a concomitant factor in pulmonary gangrene. The differential diagnosis becomes particularly difficult if putrid bronchial catarrh is present in connection with bronchiectasis; the constancy of abnormal percussion sounds and rather insignificant disturbances of the general condition speak in favor of bronchiectasis. In gangrenous inflammation of the upper respiratory passages, especially of the pharynx, the nose and its accessory cavities, one finds, aside from the putrid smell of the exhaled air, other symptoms pointing to the organ affected in particular, and the grave symptoms of affection of the lungs are absent; one can sometimes ascertain that only the air exhaled from one nostril has a putrid smell.

Prognosis. While a gangrenous process in a circumscribed territory of the lung may, in rare cases, come to a standstill, be encapsulated, and end in permanent recovery (if occurring at all, most commonly in cattle and swine), the process is, as a rule, progressive in character; sepsis develops and a fatal termination occurs. The prognosis is therefore, in general, unfavorable, and the more so when gangrene is established in a previously inflamed lung or as a complication in a grave general infection.

Treatment. One might try inhalations of finely atomized dis-*infectant solutions, or intratracheal injections, the latter after* a preliminary tracheotomy; it may exert a favorable influeuce upon the upper respiratory passages and may diminish the fetid smell (see pages 528 and 572). A circumscribed gangrenous focus may possibly be removed by pneumotomy (Pansini, O. M., 1907, 289).

Further therapeutic measures consist in strengthening and nourishing the patient, in the regulation of the cardiac action, combating hyperpyrexia, and ameliorating cough aud diarrhea.

As a matter of prophylaxis, cases where it is advisable to guard against the development of gangrene, where the exhaled air becomes fetid, should receive inhalation-sprays, or still better, intratracheal injections of some disinfectant solution (see pages 572 and 612). One must always guard against aspiration in the administration of medicine, and therefore one should entirely avoid drenching if disturbances of deglutition exist.

11. Chronic Interstitial Pneumonia. Pneumonia interstitialis chronica.

(*Pneumonia indurativa, Sclerosis pulmonum, Cirrhosis pulmonum.*)

Etiology. Chronic interstitial pneumonia occurs very rarely in horses and cattle as a primary disease. Among these must be mentioned particularly what has been described by Dieckerhoff, and later by Bang, as lardaceous pneumonia in older horses; it appears to develop after an infection. (Bang expressly states that the disease has nothing to do with tuberculosis, but Rabe claims to have found tubercle bacilli in a case of this kind.) A disease, in some respects quite similar to that mentioned, has been described by Grüter, a pneumonia developed in connection with bronchitis scleroticans in horses. Pulmonary tuberculosis, which is the basis of tuberculous arthropathy in dogs, also manifests itself as a subchronic or pronouncedly chronic induration of the lungs. The lungs contain tubercle bacilli. A further cause of chronic pneumonia may be the inhalation of dustlike bodies (fragments of plants, silicon-, lime- or coal-dust), which are absorbed from the bronchi into the interstitial pulmonary tissue, and there produce a usually circumscribed, occasionally, however, a more extensive, chronic inflammatory process. (Pneumoconiosis.) Larger foreign bodies which have penetrated into the lungs from the air passages or from the fore-stomach may exceptionally produce chronic interstitial pneumonia. Schmidt observed a traumatic inflammation of the stomach, diaphragm and lungs in a horse with bronchiectasis.

Vansteenberghe & Grisez claim, on the basis of their animal experiments, that pulmonary anthracosis is not caused by the inhalation of dust-like foreign bodies, but by the absorption of dust-like bodies, which are swallowed and only secondarily brought from the intestines into the pulmonary vessels. Numerous experiments which were stimulated by the above statement have, however, proved that the dust-like foreign bodies in natural affection do get into the lungs by inhalation; anthracosis of intestinal origin is observed only exceptionally and then it does not reach a high degree (Lüttschwager).

As a rule, the disease is a secondary affection, seen particularly after bronchial catarrh. When existing for a long time, this leads to the new formation of connective tissue in the bronchial wall and finally spreads to the neighboring interlobular connective tissue. Purulent catarrh of the bronchi, giving rise to chronic pneumonia, occurs frequently in cattle, hogs and sheep. Polyadenomatous proliferations in sheep (Prösch) and cats (Ball) may have a similar origin, but they are rather to be looked upon as true tumors (q. v.). The process arises not uncommonly from atelectatic foci (see page 586). In protracted cases of acute pneumonia a progressing inflammation in the connective tissue is also seen, surrounding the affected

lobules. This termination is observed particularly after catarrhal pneumonia, more rarely after croupous pneumonia, where, if seen at all, it occurs usually in old debilitated animals. An inflammatory new formation of connective tissue also occurs in the neighborhood of encapsulated abscesses, putrid or caseous foci, parasites, tumors, etc. Chronic pneumonia also occurs in the course of chronic pleuritis. Certain chronic infectious diseases also always finally lead, in their localization in the lungs, to chronic connective tissue inflammation.

Anatomical Changes. The affected pulmonary portion appears firmer, tougher, more tenacious, it contains less air, the cut surface shows a few cicatrices or cicatrixlike stripes between the air-containing lobules, or a whole portion of lung may be changed into an almost homogenous, firm, airless tissue, in which one can see only here and there an air-containing lobule. The shrunken portions of lung sometimes contain caseous foci and abscesses. In the lung of a cow, Joest found foci produced by chronic ossifying inflammation; Bugge saw, also in a cow, foci which had been caused by deposits of lime.

In lardaceous pneumonia of horses the upper portions of the lungs appear uniformly grayish-white or yellowish-gray and lardaceous. They contain little air and are, consequently, firm. The lower and inferior portions usually show only a few nodules and are otherwise similar. Microscopically, we find proliferation of alveolar epithelia, much new formation of connective tissue, compression of the alveoli; the histologic picture has some similarity to that of carcinoma. There is, on the other hand, some similarity between lardaceous pneumonia of horses and multiple adenoma of sheep (see tumors of the lungs), and also between focal chronic pneumonia following bronchitis scleroticans of the horse. However, in this form of chronic pneumonia, inflammatory new formation of tissue is found, particularly in the peribronchial tissue, along the blood vessels and in the interalveolar tissue (bronchopneumonia fibroplastica, Grüter). In this latter disease lardaceous-looking foci are also generally seen which, however, grow at most up to the size of a fist.

In pneumoconiosis one sees in the lungs either fibrous-calcareous nodules or streaky cicatricial bands, or on the contrary, the picture of a fibrous bronchopneumonia; after coal-dust inhalation slate-black spots are also observed. The anatomical picture usually includes the signs of chronic bronchial catarrh and chronic pleurisy.

Symptoms. The respiratory surface of the lungs becomes diminished and the expansion on inspiration is impeded in consequence of the new formation and subsequent contraction of the connective tissue. Hence, difficult respiration is noted in cases of a somewhat more extensive affection; it is partic-

ularly noticeable during work and leads to rapid tiring out of the animal. The signs of chronic bronchial catarrh are also usually present, such as a dull, weak cough, scanty, usually sharp (dry) râles, or purring or whistling sounds, etc. They are often noticeable only after deep inspiration, after exercise, after coughing, or after one has previously closed the nostrils of the animal. Disturbances of nutrition become marked later on.

On careful percussion, particularly on corresponding places of the two sides of the thorax, one can usually make out a more or less distinct dullness, occasionally tympanitic sounds over the anterior and inferior portion, as a rule behind or below the scapula. The vesicular breathing is weakened in the area of dullness or is entirely absent; in other cases one may have, on the contrary, bronchial breathing, or intensified, drawn respiratory sounds. In extensive shrinking of the pulmonary tissue many pulmonary capillaries become obliterated; consequently the second pulmonary sound is intensified.

The disease runs its course without fever, and this is important as a point of differential diagnosis with reference to acute pulmonary consolidation. However, bronchial catarrh, which is scarcely ever absent, occasionally leads to a transitory elevation of temperature.

Lardaceous pneumonia of horses at first leads to diminished appetite, listlessness, cough, emaciation, and to gradually increasing acceleration and difficulty of respiration, with the characteristics of broken-winded trouble. Only after several weeks does percussion reveal, particularly in the upper portions of the thorax, more or less distinct dullness and diminution or absence of the vesicular breathing sounds, and the presence of râles. Bang believes that a chronic alveolar pulmonary emphysema is probably always formed. Fever and acceleration of the pulse come on only after the disease has lasted a long time or after very severe service. The course of the affection is insidious and slow and difficulties in respiration only after one-half to one year, reach such a high degree that the animals can not be used for work.

In bronchopneumonia fibroplastica of horses Grüter observed frequent, strong cough, scanty nasal discharge, accelerated respiration without special use of the muscles of the flanks, unchanged percussion sounds, normal boundaries of the heart and of the lungs.

Diagnosis. When dealing with a very slowly progressing pulmonary affection, one must always think of chronic interstitial pneumonia; however, it can only comparatively rarely be diagnosticated definitely. The greatest difficulties in differential diagnosis are offered by echinococcus diseases of the lungs and by pulmonary tumors. The diagnosis is comparatively easiest if the chronic pulmonary disease comes on after the symptoms of acute croupous or catarrhal pneumonia or acute pleurisy have passed off. In cattle and also in other animals it is important to exclude tuberculosis; this can, of course, usually be done by a tuberculin test.

Treatment is absolutely non-promising, hence all efforts must be directed toward sparing the animal and promoting a good nutrition, which may retard the further development of the pathologic process. The improvement of the bronchitis will improve the condition of the patient. One might try fibrolysin (for large animals every second to fourth day, one gm., dogs 0.2 gm.; or 10 cc. and 2 cc. respectively of the prepared solution subcutaneously). Animals whose meat is used for human consumption should be slaughtered as soon as their nutritive condition has improved.

12. Echinococcus in the Lungs. Echinococcosis pulmonum.

Occurrence. Echinococcosis in general, and pulmonary echinococcosis in particular, occurs everywhere. Its frequency, however, varies within wide limits in different parts. Most commonly affected are ruminants and hogs; a few cysts are sometimes found in the lungs of horses. (For more details on the frequency of echinococcosis, see the chapter on echinococcosis of the liver.)

Etiology. Echinococci are the cysticerci of the dog parasite Tænia echinococcus, the oncospheres of which are probably carried by the blood current from the intestines into the lungs; they change into cysts in the pulmonary tissues; these cysts grow very slowly in size. Infection occurs by swallowing the ova of Tænia echinococcus with food or water (on the developmental history and the various forms of echinococcus, see echinococcosis of the liver).

Anatomical Changes. In some cases, the lungs contain only a few echinococcus cysts, in others these occur in so great numbers that they cannot be counted, so that the weight of the organ may have become increased considerably (in a case reported by Findeisen, the lungs of a steer weighed seventy-eight pounds). The surface of the lungs then becomes uneven, nodular, and the palpating hand feels the cysticerci in their interior, surrounded by a connective tissue capsule and containing a clear serous fluid. These cysts may be felt as firm, tense, globular nodules, up to the size of a fist. If the cysts are cut into they discharge a serous fluid, and the elastic membrane forming the interior surface can then be peeled off easily from the external capsule. The cysts are sometimes provided with scolices, or they may frequently be sterile, or exceptionally also contain daughter cysts. Small, dry, caseous nodules or mortarlike masses are also often present in foci, which look similar to caseous tubercles, bnt in which the microscope shows sometimes folded parts of the chitinous membrane, occasionally also hooklets. The pulmonary tissue in the immediate neighborhood of the cysts appears compressed, flaccid, either entirely void of air, or containing very little of it. Echinococcus

multilocularis has once been found in the lung of a steer. Other cysticerci are usually found in the other organs, particularly in the liver.

Symptoms. Only if the invasion is considerable, does the disease manifest itself by difficult respiration, which develops very insidiously, and which, therefore, remains unnoticed for a long time and very rarely takes on a threatening character. With such difficulties of respiration, a cough appears; it is frequently heard after short intervals, usually weak, dull, sometimes even hardly audible. Difficulties of respiration and cough may suddenly increase without any apparent cause (rupture of a cyst into a bronchus or into the pleural cavity). Sometimes exhaustion and overheating bring about an aggravation of the condition. In case of rupture of a cyst, a watery fluid is sometimes discharged through the nose, in which shreds of the chitinous membrane of the ruptured echinococcus can be seen (Bolz). Sudden aggravations of this sort may be due simply to great fatigue or to overheating.

The percussion sound remains normal if only small cysts are present or if the cysts are limited to the more central pulmonary portion. If they are more numerous and larger, circumscribed areas of dullness are found in various portions of the thorax. The percussion sound is more rarely tympanitic and the cracked-pot sound may be heard in animals with a more yielding thorax, because cysts may be crowding each other or may be situated near the pulmonary surface. Metallic sounds may possibly be heard over cysts which have become empty. The areas of dullness exhibit diminution or absence of vesicular breathing sounds. Also whistling, purring and other noises. A peculiar noise called "quurksen" by Harms is characteristic for the disease, which noise is claimed to be identical with the sound elicited on rolling and pressing a lung containing echinococcus cysts. Since it is impossible that a noise arises in the cyst synchronously with respiration, such noises must arise in the bronchi. They are simply catarrhal noises and cannot be characteristic of echinococcus of the lung.

The general condition of nutrition of the animals remains satisfactory for a long time. However, when the disease of the lungs reaches a high degree, and when it is associated with echinococcus of the liver, the animals suffer later on in their nutrition. The affection takes an afebrile course.

Diagnosis. Only severe cases can be diagnosticated with more or less probability, especially in cattle. One of the most important points to be observed is the presence of severe respiratory difficulties, while the general nutritive condition of the animals shows only very insignificant disturbances. A concomitant increase in the size of the liver must direct suspicion

toward echinococcus infection. In special cases one might make use of the complement-fixation test which has been found reliable in animals by Weinberg and Vieillard; in man by Putzu. Cough is more powerful, frequent and convulsive—at least in the beginning of pulmonary tuberculosis; the temperature is elevated. There are also symptoms of a more or less exteusive bronchial catarrh and the nutrition of the animals suffers at a comparatively early stage. Pulmonary tuberculosis is distinguished by a more extensive dullness, by bronchial breathing and by a more rapid development which is accompanied by fever.

Treatment. There is no successful treatment of the disease; hence early slaughter of the animals is advisable, because otherwise their value is decreased in consequence of emaciation which will come on.

Other Parasites in the Lungs.

Distomatosis of the lungs is frequently seen in cattle, rarely in sheep, exceptionally only in hogs, buffaloes, horses, asses and dromedaries. The invasion of the lungs is brought about in such a manner that hepatic distomas get into the hepatic and from there into the pulmonary vessels. Globular cysts from walnut to apple size are then formed in the pulmonary tissue, preferably in the posterior cutaneous lobe, which have a fibrous, sometimes calcified capsule, and may, in sheep or young cattle, be surrounded by hemorrhagically infiltrated tissue. In the yellowish or olive green contents of the cysts the ova of distoma are found and occasionally one, exceptionally two of the liver flukes.

Lung distomatosis usually does not lead to any clinical symptoms; occasionally there may be cough, possibly also a circumscribed dullness on percussion, also râles. Emaciation occasionally occurs, but it is then probably due to a simultaneous distomatosis of the liver.

In Eastern Asia and in the United States, the Paragonimus westermanni (Distomum pulmonale) is found enclosed in cysts, frequently in hogs, less often dogs and cats.

The immigration of the larvæ of Paragonimus takes place similarly to that of Ascaris larvæ (see page 356), whereby some of the larvæ go astray and may be carried to the brain. Kitt saw in an island tiger in the Zoological Garden at Munich lardaceous, indurated, diffuse nodules the size of a pea and sometimes dark red in color, produced by this parasite. Upon cross section the fluke could be seen as a gelatinous body. Throughout the tissue of the lung the eggs of the parasite were found in large groups as well as disseminated, and embedded in catarrhal exudate. The animals cough and occasionally have bloody expectoration which contains eggs of the distomum. For the purpose of prophylaxis, the affected lungs must be destroyed by means of sulphuric acid.

Neumann, and also Alessandrini, found cysts containing Dithyridium variabile in the lung of a hen. The parasite is probably the larval form of a taenia.

Lung pentastomosis caused by Pentastomum denticulatum or Linguatula denticulata has been observed in a few cases, viz., in the goat

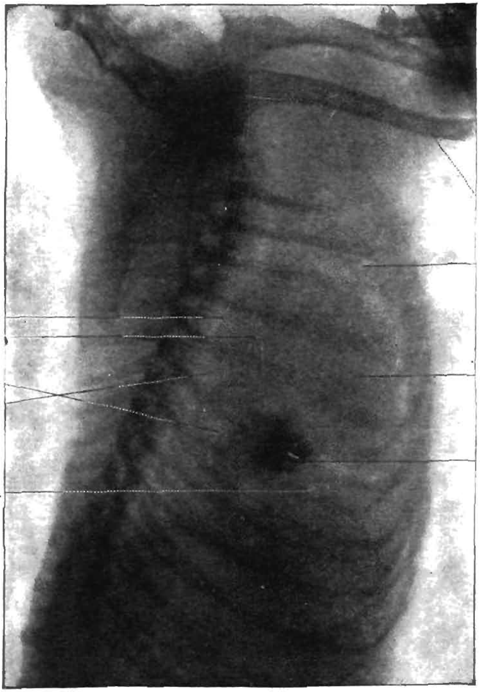

Plate III. Enlargement of tuberculous mediastinal glands in a dog. a. Mediastinal lymph-gland which takes up almost the whole portion of the thorax situated above and behind the heart, with the exception of a small strip (c); b. trachea with its main branches; c. and d. pulmonary tissue filled with air; e. heart; f. apex of the diaphragm with the liver (indistinct on account of respiratory movements); g. humerus and behind it the shadow of the muscles (Röntgenograph).

(Gerlach, v. Ratz); in the roe (v. Ratz), and in cattle (Hermann, Lungwitz). The larvæ of pentastomum burrow canals into the pulmonary tissue and frequently also into the pleuræ; numerous hemorrhagic foci are formed thereby and the animals may die from cachexia (v. Ratz).

Worm-nodules and **cysticerci**, sometimes seen in the lungs of horses and cattle, are without clinical significance.

According to the observations of Angeloff, 92.5% of the gray, translucent nodules in the lungs of horses are caused by Sclerostomum (Strongylus) larvæ. Pérard and Descazeaux found, in the peribronchial nodules of horses, nematode larvæ which they think are identical with the larvæ of Hebronema megastoma and the larvæ found in summer sores. (Concerning the occurrence of Cysticerus tenuicollis see page 991.)

13. Neoplasms of the Lungs. Tumores pulmonum.

Tumors of the lungs are not very rare; they are, however, less important from a clinical standpoint, because it is very difficult to diagnosticate them. Most frequent are carcinomata and adenocarcinomata (especially in dogs), then sarcomata and melanosarcomata (in horses) as a rule as metastatic neoplasms. Fibromata, chondromata, lipomata, osteomata, dermoid cysts are occasionally encountered. Botryomycomata are sometimes found in horses.

The symptoms which might be detected in the living animals are on the whole very indefinite in nature, because usually they refer only to a diminution of the respiratory surface; possibly also to an infiltration of parts of the pulmonary parenchyma with bronchial catarrh; the clinical picture then may be similar to chronic interstitial pneumonia. Numerous disseminated neoplasms (in Froehner's case in a horse, sarcomata) may lead to an excessive emphysema of the lungs with pronounced dyspnea. Sometimes tumors may press upon the heart or upon the larger bloodvessels and may thus bring about congestion in the territory of the jugular vein with hydropic symptoms (see compression of the heart, Vol. I), or there may be compression of a larger bronchus. Very gradually increasing dullness, eventually in combination with bronchial breathing sounds, confined to a certain portion of the thorax, may be observed and may give some information concerning an existing dyspnea. Intrathoracic tumors in smaller animals may be recognized easily in the Roentgenogram and even in larger animals if they are tolerably large and situated back of the shoulder.

However, a diagnosis of tumor can be made with a good deal of probability only if a primary neoplasm can be detected in the body, if the pulmonary affection can be referred to it and especially if a gradual enlargement of the axillary glands and those at the aperture of the thorax can be observed. Metastases in the lungs are particularly frequent in carcinoma of the thyroid in dogs.

Solitary pulmonary tumors may possibly be removed by operative procedure.

Proliferation of the alveolar epithelia in the smallest bronchi leads, frequently in sheep, not uncommonly in adult cats, to the formation of nodules of the size of a hazelnut to an apple. They are grayish-white

to reddish-gray in color, of homogeneous lardaceous consistency and occasionally become confluent to form larger tumors. These formations have been called "multiple adenomata" by Eber and Prösch, and "polyadenomata bronchiale" by Ball. They appear to arise in connection with a bronchopneumonia in sheep (Prösch) and they do not seem to lead to any disturbance of health. Ball, however, saw in cats accelerated and difficult respiration cough, a gradual increase of the size of the chest (emphysema of the lungs) without any disturbance of the general condition of nutrition (on bronchopneumonia fibroplastica, see page 708).

SECTION V.

DISEASES OF THE PLEURA.

1. Inflammation of the Pleura. Pleuritis.

Occurrence. Pleuritis is a common disease of domestic animals. It usually occurs as a secondary affection, especially after pneumonia, more rarely as a primary disease. In horses, which are most frequently affected, pleuritis develops preferably after equine influenza; in ruminants as part of the clinical picture of a hemorrhagic septicema, in cattle also after pneumonia and in its chronic form on the basis of tuberculous infection. According to Cadéac, serofibrinous pleuritis in dogs is tuberculous in nature in nine cases out of ten. The cases of pleuritis which the authors have seen in dogs almost always developed in consequence of tuberculosis. In fowls, pleuritis is said to occur frequently in combination with peritonitis (Guittard).

Etiology. The true cause of a pleuritis is generally an infection, while external influences only play a rôle as exciting or predisposing causes. The disease is often found in septicemic affections due to the bacillus bipolaris. Aside from this microbe, there are frequently concerned the pyogenic bacteria, the tubercle bacillus, the influenza bacterium, more rarely the bacillus mallei, also, in hogs and cattle, the bacillus pyogenes; Piana saw a case of pleuritis in a dog caused by the leptothrix buccalis; the authors and others saw pleuritis in dogs due to streptothrix (actinomyces) canis. Eisenmann saw cases of pleuritis following chronic hog erysipelas. Pleuritis frequently follows contagious nasal catarrh of rabbits (see page 536.) Other bacteria than those mentioned may occasionally be the cause of pleuritis.

The invasion of bacteria which cause inflammation of the pleura occurs in a variety of ways. After perforating wounds of the thorax pyogenic bacteria usually enter the thoracic cavity and produce a purulent pleuritis. Traumatic injuries to the thorax, such as a kick, contusion, a fall upon the chest, subcutaneous fracture of the ribs, which may occur without injury to the skin, give rise to the invasion of bacteria, either directly from the contused spot or through the blood current.

All these traumatic insults may cause injuries to the lungs and may cause the invasion of bacteria from the inhaled air into the pleural cavity. More dangerous are internal injuries produced by fragments of bone or other bodies which penetrate from the esophagus or stomach into the thoracic cavity.

In the great majority of cases pleuritis develops in connection with an inflammation of the immediately adjoining organs, especially the lungs, in such a manner that the bacteria causing pneumonia get to the surface of the pleura with the lymph current or else the inflammation spreads by continuity. Pleuritis very frequently follows upon croupous pneumonia, more rarely catarrhal pneumonia; but in some epizootics almost every case of bronchopneumonia leads to pleuritis. Equine influenza is accompanied in almost every case by an intense pleuritis. In purulent pneumonia and gangrene of the lungs pleuritis may occur even without any breaking through of the pulmonary abscess or dangerous focus. Tuberculosis and pulmonary glanders, also bronchial catarrh, may furnish the starting point for pleuritis.

Diseases of the other neighboring organs more rarely lead to a secondary pleuritis. Among them are to be mentioned purulent mediastinitis in the course of strangles, rupture of an esophageal diverticulum or perforation of the thoracic portion of the esophagus by a foreign body, acute and frequent traumatic pericarditis, which occurs particularly in cattle, caries and necrosis of neighboring bones, especially the ribs and sternum, acute inflammation of the peritoneum and of the abdominal organs.

In the course of acute general infectious diseases pleuritis likewise is not uncommon. In this respect the diseases ought to be first mentioned which are caused by the bipolar bacilli (see Vol. I) which may cause pleuritis without a pneumonia existing simultaneously. In the course of pyemia, in rheumatic affections of the joints and of the other serous membranes, in variola and so forth, pleuritis occasionally makes its appearance.

Pleuritis is caused exceptionally by chemicals (bacteriotoxins, constituents of urine retained in the blood in nephritis); mechanical irritation (tumors of the pleura or neighboring organs) in very rare cases by parasitic worms, for instance Pleurocercoides Baillet, in dogs and cats (Lefèbre & Guérin). and Dithyridium elongatum (Cysticercus Bailleti), in herbivora Cysticercus tenuicollis, in dogs also ascarides which have accidentally invaded the pleural space from the bronchi (Mori).

Cold frequently plays a certain rôle, aside from the causes mentioned; its influence may be very obvious, so that it is easy to understand why cold was formerly considered the exclusive cause of primary pleuritis.

The fact deserves mention that Laborde and Trasbot succeeded in producing a serous inflammation by the intrapleural injection of very small amounts of the

serous exudate from cases of rheumatic pleuritis, while the injection of pure, indifferent fluid had no effect whatever. Duvieusart observed in sheep, shorn during the winter, numerous cases of sickness which began on the second day after shearing; some cases developed even after the animals had been brought to a warm place, proving the infectious nature of the affection. (These were evidently cases of hemorrhagic septicemia.) Immelmann's observations among lambs were probably of a similar kind.

Influences which weaken the organism, such as overexertion, long-continued railroad transportation, etc., predispose animals to infections in general and to pleurisy in particular.

If there is no disease of any other organ and no general disease which might be the cause of the pleuritis, the latter is designated as a primary pleuritis in contradistinction to secondary pleuritis, which may be referred to another primary disease.

Pathogenesis. The microorganisms which are carried to the pleura from the neighboring organs or with the blood stream, multiply, and the development of the resulting inflammatory process is proportionate in rapidity to the virulence and the number of bacteria which are carried into or upon the pleura. On the other hand, an inflammation which is due solely to mechanical or chemical irritation, progresses quite slowly. In both cases the process is transmitted by contact from the affected layer of the pleura to the opposite one. Most frequently the portions in the lower and anterior parts of the thorax are affected, in analogy to the occurrence of pleurisy in connection with inflammatory lung affections. The lower parts of the pleura are also frequently involved, in case the pleurisy commences elsewhere, because they are in constant contact with the fluid exudate.

The inflammatory process commences with a dilatation of the capillaries and loosening of the epithelial layer, which is followed by the secretion of an exudate rich in fibrin and cells; the fluid portion is at once reabsorbed by the healthy pleura, and only the fibrin is deposited on the pleural surface. A liquid cellular exudate collects in the pleural space only when large quantities of fluid have been secreted which can no longer be absorbed completely.

The lysins normally present in the body and those formed in increasing quantities under the influence of the infection gradually dissolve the solid exudate and the micro-organisms. If the irritant remains active for a long time or if the exudate is very copious, its lysis requires a long time. In the meantime young connective tissue cells and young vessels grow into the fibrin deposit and give rise to connective tissue proliferation. If most micro-organisms are not dissolved, leucocytes continue to be mixed with the fluid exudate which finally becomes purulent.

The bacterial endotoxins, which have become liberated and the products of protein disintegration are absorbed into

the circulation and then produce fever. The irritation of the nerve endings in the inflamed pleura gives rise to cough, and the stitch-like pain due to the respiratory movements of the pleural layer is responsible for rapid and superficial respiration. The gradual accumulation of a fluid exudate and the masses of fibrin, which may become thick, finally separate the layers of the pleura, after which the pain becomes less or is relieved. In consequence the increased respiration becomes slower, but in its place dyspnea develops, proportional to the amount of exudate, induced by compression and collapse of lung tissue. The blood circulation is influenced largely by the toxic action of the absorbed bacterial toxins and of the products of protein cleavage (page 651). The work of the heart is hastened by reflex action on account of the pleuritic pain of the initial stage. The fluid exudate may also contribute in causing circulatory disturbances in so far as it interferes with the diastolic expansion of the ventricles. It diminishes the negative intrathoracic pressure and impedes the flow of the blood into the ventricles by compressing the vena cava and by interfering with the respiratory excursions of the lung.

Anatomical Changes. The **acute inflammation** begins with an arterial hyperemia and the surface of the pleura simultaneously becomes rough, lusterless and dry. Blood-plasma is extravasated and also a larger or less number of white blood corpuscles; fibrin is deposited in the exudate which covers the surface like a spiderweb or veil, sometimes in somewhat thicker deposits; it is very friable and loosely adherent (pleuritis fibrinosa s. sicca). In other cases, the inflammatory process leads to the formation of an abundant serous and fibrinous exudate (pleuritis seroso-fibrinosa). The serous exudate is sometimes rather clear, yellowish or reddish, possibly greenish (pl. serosa); at other times more or less cloudy, in consequence of suspended flocculi of fibrin and cells; then the pleura is covered with thicker, friable, moist, loose, yellowish fibrinous membranes.

The amount of the exudate varies very much. In the pleura of horses one generally finds 15 to 20 liters of fluid, sometimes much less, at other times much more. (Gosier found 60 liters in one case, Holmes 155 pounds of fluid); the average amount in dogs is ½ to 5 liters; in hogs 2 to 10 liters (Friedberger & Fröhner).

The thickness of the fibrinous pseudomembrane varies between some millimeters to several centimeters. Its surface is generally uneven and villous. In cases with a very rapid course the amount of fluid exudate usually predominates, in protracted cases the amount of fibrin.

In some cases, the exudate becomes purulent (pl. purulenta s. pyothorax, s. empyema), but it may then also contain fibrin membranes which cover the affected portion of lungs with fibrino-purulent deposits. Occasionally there may be an extravasation of numerous red blood corpuscles, or the exudate assumes a more or less hemorrhagic tinge (pl. hemorrhagica),

while the entrance of saprophytic bacteria causes putrefaction and an ichorous condition of the exudate (pl. ichorosa).

Under the microscope the serous exudate shows few endothelial cells, generally in a condition of fatty degeneration, and numerous leucocytes; the fibrinous coagula contain leucocytes in a reticular matrix. The purulent exudate consists preferably of intact or fatty degenerated leucocytes; while the ichorous exudate contains much granular detritus, fat droplets and shreds of tissue. The chemical analysis of the exudate shows albuminoid substances in larger amounts; 3 to 6% in the sero-fibrinous exudate, and according to Boiteux even 6 to 8.5%.

When there is a considerable mass of fluid in the pleuritic cavity, the lungs, which are already collapsed, are further compressed in proportion to the amount of fluid present; they therefore become gradually smaller and poorer in air. Those portions of lung which are in the neighborhood of the exudate become completely void of air, flaccid and of fleshy consistency; the cut surface appears uniformly dark red (compression atelectasis, splenization). When the exudate is abundant, the mediastinal lobe in horses may undergo a twist outward and backward, which displacement, according to Dieckerhoff, always leads to death, because it prevents the absorption of the exudate. (Mathis found in a dog a complete torsion of the right posterior lobe.)

Chronic pleuritis begins with the formation of vascular granulation tissue (pleuritis granulosa), which gradually becomes fibrous and cicatricial (pl. fibrosa s. productiva) or adhesions form between the pleura costalis and pulmonalis (pl. adhaesiva).

Papillomatous granulations occasionally grow out of the tissue of the pleura; Hutyra reported a case where they were very high, branching, and so numerous that they formed a mass the size of a child's heart. Cadiot and others have seen the formation of similar granulations on a tuberculous base. Kowalewsky described a case of pleuritis ossificans in a cow.

A fluid exudate is likewise not rare and is either purulent, and in this case surrounded by a connective tissue capsule (preferably in the presence of bacillus pyogenes or of foreign bodies), or serous or hemorrhagic. In horses, peculiar cases *of serous, chronic pleuritis are observed where the serous mem*branes contain a large amount of serous or slightly purulent fluid and where the pleura is studded with thick, fibrinous deposits containing numerous small abscesses. (Streptococci pleuritis, Bang.)

Symptoms. Primary pleuritis usually begins with the non-characteristic symptoms of a general disease. The sick animals gradually lose their appetite, they become dull, their fur looks bristly, they have attacks of chills, an anxious facial expression and avoid every motion. The temperature is elevated from the start. The chilliness ceases after a few hours, but with the hand placed on the thorax one feels occasionally fibrillary

trembling of the intercostal muscle (Trasbot). The number of pulse beats is, as a rule, increased 50% and the pulse is weak. Some horses show at this stage also slight symptoms of colic and they give evidences of pain (cutting) in the chest wall. In secondary pleuritis these symptoms are preceded by those of the primary disease and they may be clouded, partially or completely, by the symptoms of the primary affection.

The respiration is markedly accelerated and superficial in every case of pleuritis; it is also usually of the abdominal type, the patients hold their ribs as quiet as possible and use the diaphragm in preference. Exceptionally, however, especially in inflammation of that portion of the pleura which lies near the upper surface of the diaphragm, breathing is of the costal type.

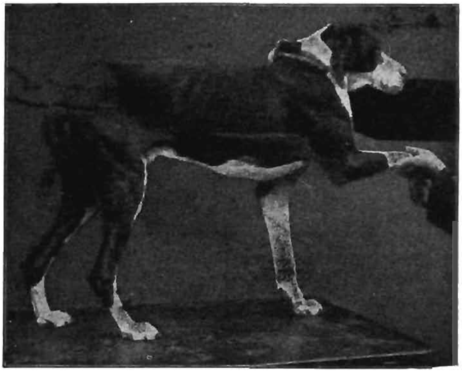

Fig. 135. Horizontal line of dullness in pleurisy with fluid exudate (Tuberculosis).

Palpation often shows an increased tenderness of the wall of the thorax, the animals try to withdraw when pressure is made upon the intercostal places, and they betray pain by crying out or groaning. The tender territory is usually behind the elbow. On palpation, and, better still, with the flat hand, one sometimes feels a trembling of the thoracic wall, which is synchronous with respiration.

Cough is not observed in all cases. When it is present, it is always weak and the animal tries to suppress it. But it may easily be brought on artificially by percussion of the thorax.

The percussion sound is normal in the beginning if the superficial portions of the lungs are not affected; in large ani-

mals a certain amount of fluid exudate may collect in the lower portion of the thorax without changing the normal percussion sound. Fibrinous membranes are almost never formed to such an extent that they will materially dull it.

The vesicular breathing is diminished. Friction sounds are frequently heard: these are sometimes soft, sometimes grating or crackling. The sounds occur synchronously with the respiratory movement, and they are sometimes heard only during inspiration, eventually only toward its end; sometimes both during inspiration and expiration. Exclusive expiratory sounds are rare. *The sounds are frequently interrupted for short periods.*

In **pleurisy with a fluid exudate** the picture so far described is altered very considerably. The respiratory movements are now usually less accelerated, but are deepened and forced in proportion to the amount of fluid present (flank breathing). During inspiration the ribs are elevated and there *is a distinct trembling of the vertebral column and of the whole* rump at the beginning of expiration. Expiration takes place in two stages and produces a groove parallel to the costal arch at the beginning of expiration; the flank depressions become prominent simultaneously, and the lumbar portion of the vertebral *column occasionally curves upward.* In very severe cases, a pumping respiration is observed. The patients also manifest other symptoms of dyspnea.

When the amount of exudate is very great, changes in the shape of the thorax may appear. In smaller animals an enlargement of the affected half of the chest is occasionally seen distinctly, while the intercostal spaces of larger animals may be flattened or even protruding. In some cases, especially in *larger animals, non-painful edemata are seen on the anterior* thorax, the lower thorax or even on the abdomen.

The tenderness on pressure of the intercostal spaces decreases or disappears entirely in the region of the fluid exudate. It persists, however, in the higher portions of the thorax if these are the seats of dry pleuritis. The painful character of the cough persists also in the further course.

Percussion shows a horizontal dullness either on one or both sides of the chest (Fig. 135), with a considerable increase in resistance. Parallel with the boundary of complete dullness and extending some centimeters upward there is some dullness, followed by a normal sound. If, however, the dullness extends over the lower half of the thorax, the percussion sound of the other half is diminished, because *the high column of the fluid* also interferes with the vibrations of the free portion of the wall of the thorax. When larger animals lie down, the area of dullness becomes diminished or it may occasionally disappear entirely and it changes also in smaller animals with

changes of position of the body (Fig. 136). On the other hand, when the exudate is encapsulated the horizontal boundary is absent and the dullness remains the same when the position of the body is changed. Only seldom is a tympanitic sound observed when the amount of fluid decreases temporarily.

Deviations from these percussion phenomena with free exudate are sometimes met with, as the author observed in a dog with a bilateral, unequally developed tuberculous pleuritis. In this case a dullness could be detected over the whole of the right wall of the thorax, while on the left side it reached only to the

Fig. 136. Horizontal line of dullness in pleurisy in sitting posture of the animal. (The same as in Fig. 135.)

height of the elbow joint, where it seemed to terminate horizontally. When the animal was placed on its back the dullness remained unchanged on the right side; its area was only reduced without entirely shifting to the back. Also after removal of the exudate from the left side, a small area of dullness remained at the lower edge of the lungs on that side, and only after the exudate was also removed from the right side did the dullness disappear entirely from the left side. With the removal of the exudate the heart beat, which had previously been fluctuating, also became normal. The exudate, which had entirely filled the right pleural cavity, not only produced a dullness covering the whole of the right side, but by displacing the mediastinum at

the bottom against the left thoracic wall also produced an unshifting dullness on the right side which remained after the removal of the exudate and which had also forced the heart out of its normal position. After the removal of the exudate from both sides the greatly distended left lung not only occupied all of the left pleural cavity, but also that part of the right pleural cavity which at the time of autopsy was not occupied by the entirely atelectatic right lung.

Auscultation shows absence of friction sounds in the area of dullness; further upward they are, however, commonly heard later on. The other respiratory sounds are likewise much diminished or absent in the area of dullness, and along the upper boundary indefinite or bronchial breathing is heard in many cases. If, however, the lung has become adherent at the onset of the disease or previously, bronchial breathing is heard also in the area of dullness. The vesicular breathing is intensified above the area of dullness and on the healthy side.

Bronchophonia shows a similar behavior to bronchial respiration. The sound due to moaning or coughing, or that produced by compression of the arytenoid cartilages is more clear and sounding at those portions of the thorax where bronchial breathing is heard. In contrast to croupous pneumonia the sound is less strong here, and seems to come from a distance. A similar behavior is noted in regard to the tracheal percussion note which is audible on the thorax.

According to Marchand & Séjournant the coin sound (signe du sou, according to Pitres) is well adapted for the recognition of pleurisy with effusion. If a coin, held firmly in an intercostal space, is struck with another coin at right angles, or better on percussion with the metal part of the percussion hammer upon the pleximeter and simultaneous auscultation is practiced over the corresponding spot on the opposite side, the percussion sound is higher and more metallic in the area of pleuritic dullness, in contrast to the more resonant, stronger, deeper and more prolonged note, or to the dull, high note of purely pneumonic infiltration. This sign depends upon transmission of the sound through the fluid exudate and is of dignostic importance only in bilateral pleurisy because in unilateral disease the sound waves are thrown back in like manner as through the lung with inflammatory infiltration; in both cases the sound is weakened. The appreciation of this sign requires much practice.

The apex beat is weakened on the diseased side and the heart sounds are duller and weaker. Toward the termination of severe cases the symptoms of cardiac weakness appear.

The temperature (Figs.137 and 138)does not give a typical curve. Great variations of temperature are usually seen in the later course of the disease. Remittent or continuous fever in less acute cases is occasionally interrupted by fever-free in-

tervals. *Very high fever is usually seen in purulent or ichorous pleuritis.*

Continuous standing during the course of the disease is frequently observed in horses. If the animals lie down exceptionally they rest on the sternum or on the healthy side, as do other animals in the beginning stage of pleurisy, in order to protect the tender portions of the thorax against pressure; after an exudate has been formed they lie on the diseased side, so that the healthy portions of lung have a chance to expand. If animals in this stage are, during examination, placed upon the healthy side, the dyspnea is at once markedly increased and the animals may even suffocate, because the healthy parts of the lungs are compressed by the body weight, and the affected portions by the exudate.

The appetite is capricious and only becomes normal after recovery has set in. If the disease lasts for a long time the animals become markedly emaciated.

The amount of urine is diminished in the beginning; the amount of chloride of sodium diminishes markedly as the exudate forms, while its absorption leads to an increase of urine and of its chloride of sodium contents (urinary crisis). Albuminuria is not uncommonly present during the course of the disease.

Complications are frequent, even if the primary diseases followed by a secondary pleurisy are left out of consideration. Very important, as a complication, is pericarditis, because it may lead to complete cardiac exhaustion. Delacroix found embolism in the pulmonary arteries in a horse which died suddenly during recovery from pleuritis; the origin of the embolism was found in a thrombotic, bent posterior vena cava. An abscess is sometimes found in the wall of the thorax in purulent and ichorous pleuritis; the abscess breaks outside, or it opens into one of the thoracic organs, into the lungs, or through a cavity of the latter into a larger bronchus, into the trachea, pharynx, mediastinum, etc.

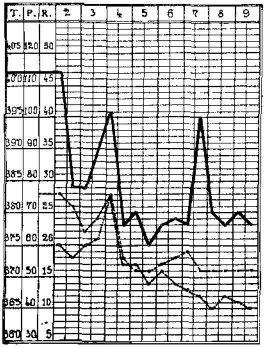

Fig. 137. Fever curve in primary *pleurisy in a horse. Recovery.*

According to Guittard, **pleuropneumonia of fowl** at first leads to listlessness, diminished appetite, retarded digestion, and after several

days to great prostration, the comb becomes pale and the respiration forced. In the further course of the disease the animals die quickly after diarrhea has set in. An exact diagnosis can only be made on postmortem examination.

In the beginning and often during its whole course, **chronic pleuritis** does not lead to very prominent symptoms, which become marked especially during exercise. If there are extensive adhesions, the vesicular breathing is less marked, but the percussion sounds remain normal. In the presence of serous or more purulent exudates in the thoracic cavity, after certain occurrences, for instance after hard work or after a cold, one observes acute exacerbations which, in addition to already exist-

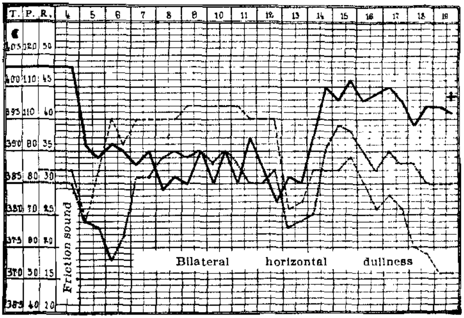

Fig. 138. Fever curve in secondary pleurisy in a horse, (following upon croupous pneumonia).

ing areas of dullness and diminution or absence of breathing sounds, lead to tenderness of the thoracic wall, marked dyspnea, dry, rough cough and moderate elevation of temperature. Displacement of the heart by the exudate leads to change of the apex beat and in the intensity of the different heart sounds in various places of the cardiac region; disturbances of circulation may also be present (stronger filling of the veins, presystolic venous pulse, edematous infiltrations).

Disturbances of nutrition appear sooner or later. The hair becomes lusterless and ruffled, the skin dry and less elastic; emaciation gradually reaches a high degree, the production of milk diminishes, the symptoms of cardiac weakness come on and the animals die exhausted and emaciated.

Course. Very mild cases of fibrinous pleurisy sometimes run a very rapid course, occasionally one of a few hours only.

The symptoms may be of a very minor character, very insignificant tenderness, soft friction sounds; otherwise nothing abnormal can be detected in the chest. These symptoms may entirely escape notice, as is proved by the well-known fact that thickening of the pleura and adhesions are not infrequently found in animals who have always been under observation and who never showed symptoms of pleurisy.

The formation of an abundant fluid exudate often requires a long period of time; sometimes, however, the pleural cavity may become filled within a few days to over one-half of its space. The increase of the exudate usually occurs periodically and is accompanied by elevation of temperature. Occasionally the exudate only increases to such an extent that life is not endangered for several months.

Pleurisy with exudate may end in complete recovery, especially when the exudate is not very fibriuous or is purely serous. In the majority of cases chronic tissue changes, such as thickening and adhesions, remain as permanent residua. Whenever the quantity of the exudate is considerable, its absorption requires several weeks and is interrupted by temporary relapses. Adhesions which have been formed may subsequently interfere with the proper movements of the lungs. Most dangerous in this respect are adhesions at the lower posterior thirds of the lungs, or the lower posterior margins. Animals with adhesions in these parts usually suffer for the rest of their lives from shortness of breath, even if their general state of nutrition is good. Adhesions of the lungs may also bring it about that the portion which is fixed in an abnormal place is unable to return to its normal place after the exudate is absorbed. The neighboring portions must then expand more extensively, the heart may be displaced toward the affected side and the thoracic wall may eventually be depressed.

Chronic pleurisy does not terminate in complete recovery. Such animals are easily tired out when worked, their nutrition is not good, they cough periodically, breathe with difficulty and the presence of an exudate in the lower portion of the thorax can be demonstrated objectively.

A fatal termination of pleurisy occurs in acute cases usually after two to three weeks, in consequence of suffocation or exhaustion. Particularly in cats, the excitement resulting from the examination may bring on an attack of suffocation and heart weakness. For this reason such animals should be handled with great care in cases where a collection in the pleural cavity is suspected (Lasserre and Lesbouyries). In some chronic cases the animals become completely prostrated and die.

Diagnosis. The only pathognomonic sign of pure fibrinous pleuritis is the friction sound heard synchronously with the respiratory movements. If, however, in the course of a disease which is usually followed by pleurisy, tenderness of the

intercostal spaces come on, one is justified in thinking of the probability of a beginning pleurisy. Rheumatism of the intercostal spaces (pleurodynia) is very rare and can be distinguished from pleurisy, even in the absence of friction sounds, on account of marked general disturbances which are present in pleurisy, by the accelerated and weak pulse, acceleration of the superficial, so-to-say trembling, respiratory movements. Affections of the ribs (fracture, periostitis, caries) are characterized by swelling and tenderness confined to the ribs, as well as by the absence of general symptoms.

Pleuritis with fluid exudate is characterized by the following clinical picture: marked horizontal dullness, often on both sides, strong increase of resistance and the absence of respiratory sounds in the area of dullness. Exploratory puncture may be necessary to clear up a doubtful case. Croupous pneumonia is differentiated by the presence of an area of dullness which is bounded upward by a curved line or which rises or falls posteriorly, and by bronchial breathing which is audible at least from time to time. The initial symptoms are also very different. The onset of pneumonia is characterized by a reddish yellow discoloration of the mucosæ, eventually by a rust-colored nasal discharge, by deep and difficult respiration and by a strong pulse; in the beginning of pleurisy the respiration is much accelerated, superficial, almost trembling; the pulse is hard and small and the hand placed on the chest wall can detect a muscular tremor. The fever curves of the two diseases differ materially. The course of the fever in pleurisy is similar to that in catarrhal pneumonia; however, a continuous area of dullness is either absent or it is shaped as in croupous pneumonia.

The difficulties increase, however, when it must be decided whether pneumonia and pleurisy exist simultaneously. The shape of the area of dullness is then not decisive, because fluid below and infiltrated lung floating on it above may give a curved boundary line of the area of dullness. One also hears bronchial breathing sounds in the floating lung especially near the upper boundary of the area of dullness, as it is often found in croupous pneumonia. The behavior of the apex beat and of the heart sounds often is decisive; a diminution in their intensity points to pleurisy. Important also is the presence of edema of the inferior thorax, which does not occur in pneumonia; also the behavior of the transmitted tracheal percussion note and of bronchophony (see page 653).

The differential diagnosis between pleurisy and pleuropneumonia in cattle may be difficult when pneumonia consolidation exists simultaneously with pleurisy. The concomitant circumstances, such as the possibility of importation of the contagion must then be considered. A suspicion of the existence of pleuropneumonia appears justified where subacute or chronic pleuritis or pleuropneumonia are found and where the possibility of a contagion cannot be excluded.

Pleuritis may be distinguished from pericarditis by the fact that in the former case the apex beat is not as much intensified and as rhythmic as in the latter case; the friction sounds in pleuritis are usually heard on one side only, they are influenced by the respiratory movements, and friction sounds and tenderness of intercostal spaces are usually found in other areas aside from that of the heart. Apex beat and heart sounds are almost absent in pericarditis with effusion; this is contrary to what is found in pleurisy.

Auamestic data and the eventual simultaneous presence of other pathologic processes deserve full consideration in establishing the particular nature of existing pleurisy, which may depend upon a purulent inflammation of neighboring or more distant organs; purulent or ichorous pleurisy sometimes occurs after penetrating wounds of the thorax. Pleurisy accompanying chronic affections of the lungs (tuberculosis, glanders, actinomycosis, lungworm disease) is usually fibrinous or serofibrinous. In tuberculosis and in cachectic processes in general it may also be hemorrhagic. Assistance in the diagnosis may be afforded in cattle and also in dogs by a tuberculin test. The character of the exudate is best ascertained by an exploratory puncture, which is perfectly void of danger and which should be performed in all doubtful cases. The fluid is examined microscopically, chemically and bacteriologically; in case a tuberculous pleurisy is suspected, it may be used for inoculation of experiment animals. Guinea pigs inoculated with tuberculous pleuritic exudate apparently free from tubercle bacilli, ofteu acquire tuberculosis. Such an exudate contains particularly mononuclear leucocytes, as does the exudate of chronic pleuritis especially in intrathoracic tumors.

The **exploratory puncture** is best made with a hypodermic syringe; its needle is pushed into the area of dullness, into the pleural cavity, best in the sixth or seventh intercostal spaces. In withdrawing the piston of the syringe, fluid often enters even if the needle has been pushed into masses of fibrin. The end of the needle may touch the lung and in this manner we may eventually gain some information about the consistency of the lungs; if the needle has been pushed into the lung parenchyma, we get pure blood or, in the presence of pneumonia, a bloody stained fluid. The fluid obtained by puncture may be used for bacterioscopic examination and for animal inoculation.

The **prognosis** depends upon the etiologic factors, the character of the exudate, the species and the age of the animals affected. Pure fibrinous pleurisy following croupous pneumonia, is not of very grave significance, but the accumulation of a fluid exudate under these and other circumstances is usually an unfavorable symptom. Cases with high fever, chills and great prostration, with rapid increase of the exudate, usually take an unfavorable course. A guarded prognosis is, however, indicated even in less tempestuous cases, because relapses are common and may lead to a fatal issue. Speedy death must be expected in purulent and ichorous pleurisy. The formation of a

hemorrhagic exudate into the pleura under marked acute symptoms also has an unfavorable significance.

In *considering* the prognosis one must also think of the sequelæ which may materially decrease the value of the animal.

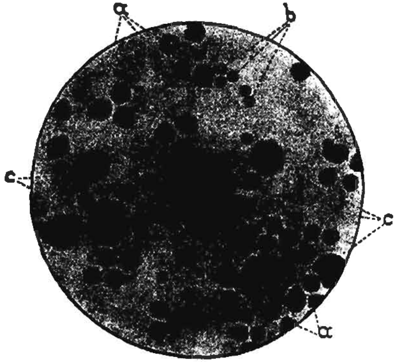

Fig. 139. Sediment of an exudate in serofibrinous pleurisy of a horse. *a*, polynuclear leucocytes; *b*, red blood-corpuscles; *c*, endothelial cells in a condition of fatty degeneration.

Adhesions usually remain after a pleurisy which has lasted three to four weeks; relapses and the formation of an area of permanent dullness exclude the possibility of complete recovery.

Treatment. The most important measures are the establishment of favorable hygienic conditions, regulation of diet and nutrition of the animal following the same principles as laid down for the treatment of croupous pneumonia (see page 666). Energetic antiphlogistic treatment is indicated in the beginning of pleurisy when friction sounds are still present. To this end the thoracic wall should be irrigated with cold water, or snow or ice should be applied to the tender region. This must be continued until the sensitiveness, the friction sound, and the high fever has subsided. Good results may be obtained in the beginning with these applications. Inunctions with irritating or acrid substances, based upon the principle of counter-irritation, appear less indicated, although they were formerly much practiced.

French veterinarians even today extensively employ the socalled revulsive treatment by irritating inunctions applied to the thorax from the start and alleged to stop the inflammation. Venesection is practiced for the same reason.

Autoserotherapy, which was introduced by Magnin, is said to have proved of value especially in the initial stage of exuda-

tive pleurisy in horses. In several cases observed by the authors, it failed to influence the course of the disease, but of seventeen horses treated in this manner by Marchal & Séjourant, all recovered. As soon as possible after an exudate has formed, thoracocentesis is practiced and a portion (10-15-50 cc.) of the removed fluid is injected under the skin without reference to the character of the exudate. If necessary the treatment is to be repeated on several consecutive days. (Mello withdraws the needle of the syringe which has been filled with the exudate only as far as the subcutis and injects the exudate at the point of puncture.)

Chiari and Januschke recommend chloride or lactate of calcium, to prevent the formation of exudate. In experiment animals these drugs prevent the development of inflammation; they are given internally (dogs 0.5-1.0 gm., horses 10-20 gm. in solution), or better subcutaneously (10-20 cc. of a 2-4% solution) for several days. In a dog with tuberculous pleurisy, and in a horse with recent pleuropneumonia these remedies did not prevent exudate, but in some horses intravenous injections of a 1% solution of calcium chloride in physiological salt solution proved successful in the hands of the authors.

In the further course, beginning on the second or third day, moist, warm applications may be made. Energetic inunctions may be serviceable in delayed absorption, viz., spirits of mustard, 6-10%, chloroform liniment (chloroform 10.0, liniment volatile 40.0). These are to be applied twice or thrice daily over the area of dullness, while more intensely acting drugs, such as cantharidin, croton oil, euphorbium ointment, etc., should rather be left alone. Priessnitz' applications may be employed two to three hours after the inunctions.

NOTE.—In human medicine applications of green soap have proved very efficient means of stimulating delayed absorption of pleuritic exudates (see Report from the Winyah Sanatorium, Drs. K. and S. von Ruck, Asheville, N. C., 1911, page 4). This should prove of value in animals also. The green soap is applied by friction over the affected side after the skin has been washed with ordinary soap and hot water. As a rule the application is made at bed time, is allowed to remain over night and repeated daily, or less frequently, depending upon the tolerance of the skin. When hyperemia occurs as an effect of irritation, this is allowed to subside before the application is repeated. In many cases the skin is tolerant enough to permit daily applications; in others marked hyperemia follows after its first use in which event the soap must be diluted (Transl.).

Narcotics are to be employed in severe tenderness of the thoracic wall and against tormenting cough. Fever requires interference only when it is very high and when the affection has set in in a very tempestuous manner; then the drugs recommended elsewhere (see page 666) are to be considered.

Diuretics are indicated in the presence of a diminished excretion of urine; powerfully acting drugs should however be avoided. Aside from those medicines which were recommended for croupous pneumonia (see page 666) the following are to be

recommended: digitalis (daily 3-7 gm. or to dogs as an infusion 1 : 100 in tablespoon doses, not to be continued longer than one week; liq. potassii acetatis (100-180.0 or 5-10.0 per diem, also in combination with digitalis); oil of turpentine (10-15.0 or 1-2.0); calomel (2-4.0 or 0.03-0.05) must be discontinued at once after diarrhea has set in; common salt. To increase absorption the following drugs are serviceable: pilocarpine (0.1-0.2 and for small animals 0.005-0.01 gm. subcutaneously); arecoline (0.08 gm. for horses subcutaneously—LeFèvre always had good results following the injection at short intervals of small doses of 0.03 gm. each), and the laxatives, especially the salts. Animals with cardiac weakness should not receive pilocarpine or arecoline.

The most reliable procedure to diminish the amount of the exudate is puncture of the thorax (thoracocentesis), an interference absolutely void of danger if performed with aseptic and general precautions. Thoracocentesis is indicated especially when there is immediate danger of suffocation and in purulent and ichorous pleurisy. There exist, on the other hand, numerous observations (Almy, Liénaux, Fröhner, Marek), which show that puncture made repeatedly and immediately after the exudate has formed may bring about a speedy amelioration even if only a small amount of fluid has been removed. The often observed favorable influence, even after the removal of a small amount of exudate, may be explained by a diminution of the pressure exerted upon the blood and lymph vessels of the pleura.

The primary affections, of course, influence materially the possibility of successful treatment in secondary pleurisy. Fröhner is inclined to believe that only those cases offer hope in which the exudate is free from bacteria.

The **puncture** is best made in the seventh intercostal space but on the right *side it may be made in the sixth space; in larger animals 3 to 5 cm. above the costal* cartilages. The instrument may be simply a hollow needle provided with a long, slender rubber tube, the free-end of which should dip into sterile water. A trochar (Billroth's trochar is particularly serviceable) is still better adapted which has the canula provided with a stop-cock which should be closed after the withdrawal of the stiletto; then a rubber tube can be fastened to the trochar. This simple procedure does, however, not always lead to success, because the exudate rarely stands under sufficient pressure to force the fluid out of the rubber tube. It is therefore frequently necessary to employ the aspirators of Dieulafoy or Potain, which will permit the aspiration of fluid out of the thorax and offer the additional advantage of preventing the entrance of air into the thoracic cavity. The entrance of bacteria into the thorax during puncture must by all means be prevented because this would lead to a more serious and more malignant inflammation.

The evacuation of the exudate must always be brought about gradually and slowly in order to avoid sudden changes of the intrathoracic pressure, as these might lead to extensive capillary hemorrhages and paralysis of the heart. The puncture must be interrupted at once if the yellowish exudate becomes hemorrhagic, if the patient is attacked by cough or if the needle touches the lung. If the pulse becomes weak the patient should receive a subcutaneous injection of caffeine or oil of camphor. Occasionally one might try intravenous injections of adrenalin (Barr). After the termination of the puncture horses may receive a subcutaneous (Boucheriou) or an intravenous (Almy) injection of 2 or 3 liters of physiologic salt solution. Fairise recommends a subsequent intrapleural injection of 1½ to 4 liters of a 1 to 1.5% solution of gelatin in order to prevent hemorrhage or a rapid

renewal of the exudate, Bouchet the introduction of air with Potain's apparatus, while Poeanaru and Falcoianu achieved a cure in three horses with two or three injections of 10 to 15 cc. of adrenalin into the pleural cavity after the exudate was withdrawn. Subsequent dribbling of the exudate into the intramuscular tissue through the puncture in the thoracic wall may be prevented by the use of a finer trocar.

The amount of exudate which may be removed by one puncture depends upon circumstances; if there is intense dyspnea, enough should be withdrawn to remove the latter (in horses 20 to 40 quarts). In cases where the amount of the exudate is moderate and where there is no immediate danger of suffocation, the removal of 4 to 5 quarts in large, and of 250 to 500 cc. in small animals, is sufficient; larger amounts should be removed at one time only if repeated punctures have shown that additional amounts of fluid have not been formed. It is well known that horses can stand, without damage, the removal, at one time, of 30 to 40 or 50 quarts of fluid; however, this may exceptionally lead to the death of the animal, either in consequence of paralysis of the heart (Trasbot, authors' observation) or on account of extensive capillary hemorrhage (authors' observation). These circumstances, as well as the fact that withdrawn fluid may reaccumulate in a short time and so weaken the patient considerably, let it appear advisable not to withdraw very large amounts of fluid at one time.

Simple puncture is not sufficient in purulent or ichorous pleurisy and it is usually not even successful if the thoracic cavity subsequently is irrigated repeatedly with a disinfecting solution (boracic acid 2-4%; thymol, 0.1%; acetate of lead, 1-2%). Bergeon succeeded in a cow by repeated irrigations with the following solution: tincture of iodine, 60.0, iodide of potash, 20.0, boiled water, 10,000 cc. Ordinarily the only procedure that holds out any promise of success is to open the thoracic cavity, and the resection of ribs, followed by cleansing of the chest cavity (Cadéac, Poinot and others).

2. Dropsy of the Thoracic Cavity. Hydrothorax.

Hydrothorax occurs as a part of the clinical picture of certain organic and general diseases and consists in the collection of a serous fluid in the thorax without inflammatory changes in the pleura.

Etiology. Hydrothorax is usually caused by a congestion in the vena cava and its branches. It is therefore observed in chronic diseases of the lungs; still more frequently in chronic cardiac diseases and is usually associated with ascites, hydropericardium and anasarca. The affection is similarly developed in compression of the venous trunks (compare compression of the heart in Vol. I).

Hydrothorax in other cases depends upon general anemia, hydremia, or indirectly upon a long-standing exhausting disease (glanders, renal disease, echinococcus, carcinomatosis, etc.). The effect of substances which are irritating to the capillary walls may play a rôle in some cases.

Joest observed a bilateral chylothorax in a dog, resulting from the compression of the thoracic duct by a neoplasm of the thymus gland.

Anatomical Changes. The transudate in the thoracic cavity appears light, or reddish yellow, perfectly clear or slightly

turbid; it may contain very fine flocculi of fibrin, occasionally also numerous red blood corpuscles. It contains a considerable *amount of albumin; however, the amount of the latter and the* specific gravity are less than that of the blood serum. One usually sees few formed elements under the microscope (desquamated, swollen epithelia, a few lymph corpuscles and erythrocytes and some cells in a condition of fatty degeneration). *The lungs are compressed in proportion to the amount* of the transudate; the pleuræ either show no changes at all, or are somewhat thickened or cloudy. Congestion is usually present.

Symptoms. Hydrothorax usually develops uniformly on both sides and the signs pointing to a collection of fluid in the thoracic cavity (see page 731) are found bilaterally. The other signs, especially those of compression of the lungs, are the same as in serous pleuritis; they develop, however, somewhat *more slowly, without fever and without tenderness of the thoracic wall.* The serous fluid easily changes its place and the area of dullness changes with changes in position of the patient; the upper boundary always remains horizontal. The lower portions of the thorax appear wider in the presence of an abundant transudate and the movement of the ribs also requires the action of the accessory respiratory muscles.

The **diagnosis** depends upon the detection of the primary disease, the presence of other hydropic conditions, the protracted and variable course of the affection. Exploratory puncture furnishes further proof; the fluid is usually only slightly turbid, the specific gravity below 1016, the sediment contains few white blood cells and does not cause cloudiness, or only slight cloudiness on the addition of one drop of the fluid to a highly diluted acetic acid (1 drop to 100 cc. of water), in contrast to the smoky, milky or ring-shaped cloudiness in case of exudates. Chaleur and Labasque removed the transudate of a horse suffering from compression of the vena cava due to a melanotic tumor, and found black, sand-like masses of pigment.

Treatment. The amelioration or aggravation of hydrothorax goes hand in hand with the primary disease toward which the treatment has to be directed. If suffocation threatens, puncture may be resorted to. It may afford temporary relief.

3. Collection of Air in the Pleural Cavity. Pneumothorax.

By pneumothorax in the limited sense of the term is meant a pathological condition, when atmospheric air gets between the parietal and visceral layers of the pleura and causes col-

lapse or compression of the lungs. Those rare cases must also be included where other gases get into the pleural cavity.

Etiology. Air is enabled to enter into the pleural cavity in consequence of penetrating traumatic injury to the chest wall, on account of the negative pressure in the chest cavity. The lungs then collapse in consequence of their elasticity. Pneumothorax is similarly brought about when a foreign body penetrates into the diaphragm from the omasum; so that the gases in the latter can get into the pleural cavity. Pneumothorax is also frequently observed in consequence of disease of the lungs. Injury to the lungs may occur in penetrating trauma to the thoracic wall, in subcutaneous fracture of the ribs, or through a foreign body aspirated into the air passages. The possibility of rupture of the superficial strata of the lungs also exists when the air in the lungs is suddenly placed under high pressure (cough, violent action of the abdominal walls, drawing of heavy loads, vomiting, great efforts, continuous bellowing, falling, sudden pressure upon the thorax, etc.). Purulent or ichorous pulmonary foci communicating with bronchi, occasionally echinococcus cysts (Deupser, Sperling) may break into the pleural cavity. Perforation of the pharynx may in very rare cases lead to pneumothorax.

Pathogenesis. The amount of air or gas which enters depends upon the character of the opening. If the latter is not closed (open pneumothorax) enough air enters to bring about a degree of collapse of the lungs which is seen in cadavers after opening of the thorax. In 2 to 2½ hours, the air left in the prolapsed lung is also absorbed and the latter may become entirely void of air. If, however, the opening is of such type that it will open during inspiration and close during expiration (valvular pneumothorax), then the air pumped in on inspiration will rapidly compress the lungs and expand the wall of the thorax. If the opening closes sooner or later (closed pneumothorax) the lungs will also collapse proportionately to the amount of air entered. But on account of the closure of the opening they will not become compressed, the air is uniformly distributed around the external and lower portions of the lungs, and can be absorbed later on.

With the air streaming in through the wall of the thorax or from the air passages and always with air coming from neighboring organs, bacteria get into the pleural cavity and may lead to fibrinous, more usually to purulent, pleuritis.

Anatomical Changes. Air does not stream outward or inward in open pneumothorax after opening of the thoracic wall; it streams occasionally into the chest cavity in closed pneumothorax and streams out with a hissing sound in valve pneumo-

thorax; it may then move a flame held before the opening. If the trachea had been closed air-tight before an injury to the lung, on opening the chest cavity, one can easily recognize the collapse of the lungs as it occurred during life. We also usually find the signs of serofibrinous (hydropneumothorax) or purulent pleuritis (pyopneumothorax). The changes are usually found in one, rarely in both pleural cavities.

Symptoms. The symptoms of pneumothorax either set in suddenly or they develop very gradually, according to whether air got into the pleural cavity suddenly or gradually. More or less intense dyspnea develops in consequence of the displacement and collapse of the lung. It often increases gradually and may lead to attacks of suffocation and to a fatal termination, within one-half to one day. Rapid deterioration is particularly seen after bilateral pneumothorax or after one due to the breaking through of an abscess or of a gangrenous focus. Slow ingress of air in other cases leads to much less violent symptoms; these may, in fact, be so slight that they are overlooked and the pneumothorax is only discovered during a physical examination.

The shape of the thorax in valvular pneumothorax may be so changed that the diseased half is more prominent, while in other forms the healthy side or both halves may become prominent. Tenderness on pressure upon the intercostal spaces points to pleuritis or to fracture of the ribs.

In valvular pneumothorax, the percussion sound is atympanitic, loud, and possibly somewhat weakened, in consequence of increased tension of the chest wall. In open pneumothorax, the sound may be tympanitic in all species of domestic animals, in the presence of a sufficiently large opening in the thorax wall; if, however, pneumothorax is open toward the air passages and if the opening can not be large, a tympanitic sound is only heard in small animals, because in them only the small column of air necessary for the production of tympanitic sound will be present under these conditions. In open pneumothorax one commonly also hears the noise of the cracked pot. The percussion sound of a closed pneumothorax, in total or considerable pulmonary collapse, remains atympanitic in large animals; it becomes, however, distinctly tympanitic in small animals, and also in large animals if conditions have brought about the formation of a small column of air.

One usually hears a metallic sound on ordinary percussion, frequently only on auscultating or on percussion with two hard objects; this is, as a rule, audible over the whole of one-half of the thorax. If adhesions have previously been formed, or if rapid closnre of the opening has permitted the entrance of only an inconsiderable amount of air, the metallic sound is heard over a limited area only. When there is an open communication of the pleural cavity with the air passages, one hears the metallic sound, even at a distance, in smaller animals after opening their

mouth. The percussion sound of the chest may, posteriorly, on the affected side be followed up to the costal arch; the same occurs, however, on the healthy side in consequence of a compensatory distention of the lung on the healthy side.

After the development of pleurisy with effusion, one can demonstrate dullness to a variable extent with an upward horizontal boundary line; here there is no metallic sound.

Auscultation demonstrates conditions which vary from case to case. Sometimes one hears no breathing sounds at all in consequence of intense collapse of the lungs; as a rule, however, the breathing sounds penetrate through a not overly thick column of air. One then hears bronchial, possibly amphoric breathing; usually, however, only a metallic sound. If there is fluid and air between the layers of the pleura, one hears metallic splashing with every respiratory movement, which becomes still more intense in sudden movements or in shaking of the animals. In exceptional cases, one sometimes hears dripping sounds.

The heart beat is not felt at all on the affected side, or only faintly; the heart sounds are occasionally accompanied by a metallic note.

Course and Prognosis. The described symptoms usually last only a short time, especially if pneumothorax follows an already existing pneumonia or pleurisy; however, even in animals which were previously well, pleurisy usually develops soon and brings about a fatal issue. Pneumothorax from injury or rupture of a healthy lung ends in recovery somewhat more frequently; one due to penetrating injury of the thoracic wall rarely, i. e., when infection of the pleura has not occurred. The air is absorbed in such cases after closure of the injury, and the lungs again expand.

Diagnosis. Characteristic for pneumothorax is the metallic sound present over a large portion of the thorax and brought out by percussion and auscultation. When lung cavities are present, tympanitic and occasionally also metallic sounds are present only in circumscribed places, and they are usually surrounded by an area of dullness. Diaphragmatic hernia might give rise to errors in diagnosis, if parts of the stomach or loops of intestines have prolapsed into the thorax. But the tympanitic or metallic sound which may be present in the posterior and inferior portions of the thorax reaches a high intensity; its pitch varies with the peristaltic movements and, if these are arrested on account of incarceration, the symptoms of intestinal occlusion are present. The character of the fluid which might *be present can only be ascertained by an exploratory puncture.*

Treatment. In closed pneumothorax, the air ought to be removed. An ordinary trochar is not well adapted for this purpose, because more air might be let in by it. Therefore, one has to use a trochar with a stopcock or, still better, a Dieulafoy

aspiration apparatus. In the absence of such devices, a simple trochar or hollow needle may still be serviceable if one attaches to it a rubber tube with a clamp. In pneumothorax which opens outward the opening must first be closed.

The operation recommended for purulent pleurisy might be tried in purulent pneumothorax.

Diseases of the Circulatory Organs

SECTION 1.

DISEASES OF THE PERICARDIUM.

1. Pericarditis.

Occurrence. Pericarditis is not a very prevalent disease. It occurs most frequently in cattle in the form of traumatic pericarditis, inflammations of the pericardium from other causes being much more rare, and in all probability usually tuberculous in origin. Still more rarely the disease is observed *in horses, in which it usually occurs as a sequel of croupous* pneumonia. It occurs a little more often in dogs, usually on a tuberculous basis, less often in distemper or other diseases. In hogs pericarditis is not very rare, appearing most often in swine plague, less often in erysipelas. In fowls the disease is also observed occasionally, usually as a complication of cholera and chicken plague, less often in tuberculosis.

Etiology. The occurrence of pericardial inflammation is due, *almost without exception*, to the immediate influence of infectious substances, although these often become effective only if certain predisposing conditions are also present. Searching bacteriological examinations have not been made very often with respect to pericarditis in animals, but those that have been reported, and also the mode of onset of the disease, permit the assumption that the infectious substances mentioned below are particularly concerned in the production of pericarditis. The Bac. bipolaris may be active in this manner, not only alone but also in association with other bacteria; in fact, there is hardly one of the diseases for which this bacterium is responsible that is not occasionally associated with pericarditis. Further, the pyogenic bacteria, the tubercle bacillus, the virus of swine érysipelas, chicken plague, contagious pleuro-pneumonia of cattle and of variola may also take part in the production of pericarditis.

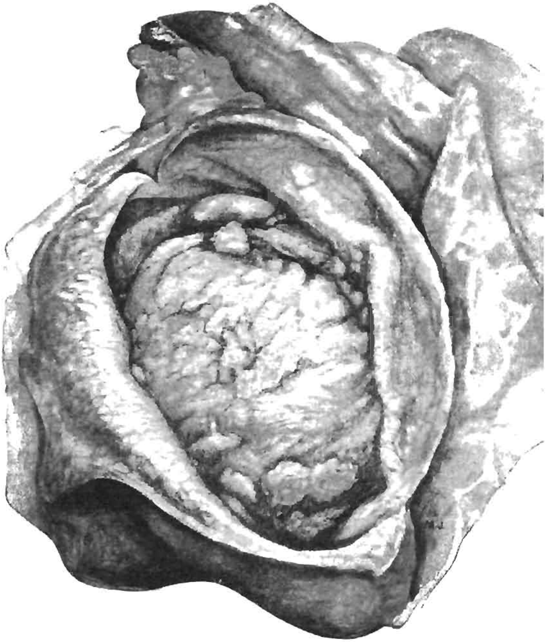

Fig. 140. Traumatic pericarditis in a cow. Massive deposits of fibrin on bo serous layers of the pericardial sac, which was greatly distended with exuda

The point of entrance of the microorganisms may either be in an injury of the thorax, or more frequently the virus reaches the pericardium by way of the blood and lymph streams. The infection is most easily explained when traumatic influences affect the pericardium, and when pyogenic or putrefactive bacteria enter simultaneously with the injuring object or pass in subsequently through the opening. Penetrating injuries of the heart region are rather rare as a whole, and likewise rarely an effect of contusions of the praecordia is exerted without penetration of the wall of the thorax, so that bacteria which have entered the blood in another manner are carried to the pericardium and then proliferate in the contused tissne. Even simple contusion of the heart region, without a penetrating injury of the wall of the thorax, may under certain conditions induce the localization in the pericardium of microorganisms which have accidentally entered the blood.

Those injuries of the pericardium are of greater frequency and of the highest importance in which foreign bodies have entered the pericardium from neighboring organs (Pericarditis traumatica). Pericarditis of such an origin occurs frequently in cattle, less often in goats and sheep and only exceptionally in the other animals. In a case observed by Henniges in a horse a needle, probably from the esophagus, entered the pericardium; in a case of Lewin's, from the stomach or duodenum; in a hog a needle came from an unknown part of the body (Teetz), or from the stomach (Nietschke), while in a dog seen by Petit the foreign body (needle) had probably passed with the blood of the posterior vena cava into the heart and had partially advanced into the pericardium after perforating the wall of the heart.

In the case reported by Borg a sewing needle 3 cm. long probably penetrated from the duodenum through the diaphragm near the apex of the heart into the pericardial sac and from there into the left ventricle. It was then carried along by the blood stream into the atrioventricular opening, where it penetrated the mitral valve and the wall of the ventricle. In ruminants foreign bodies which have been swallowed usually pass from the second stomach toward the pericardium. The anterior surface of the reticulum is separated from the pericardium only by the thin diaphragm which is easily perforated even by comparatively short foreign bodies. In this respect needles are most dangerous because, after having perforated the mucosa of the reticulum they are constantly pushed farther by the repeated contractions of the muscles of the reticulum until the points reach the diaphragm; then the contractions of the latter aid in pushing the needles against the pericardium and in finally entering it. Of similar importance are other rigid bodies which are more or less pointed at both ends, especially thin wire (according to Collas parts of the wire which has latterly been

used for packing pressed hay are responsible relatively often), nails, knife blades, etc. Together with the foreign bodies bacteria usually enter the pericardium as a matter of course.

The intermediation of infection by the blood- and lymph-streams must be assumed in those cases in which the affection develops in association with diseases of neighboring organs or in the course of a generalized acute infectious disease. Aside from inflammations of the heart muscle or the endocardium which easily pass over into the pericardium which is connected with the intramuscular connective tissue, pericarditis occurs preferably as a sequel to pleurisy, pneumonia, pulmonary or pleural tuberculosis and is produced in these cases by the virus of the related diseases. Pericarditis may also develop directly, by continuity, after perforation of the gullet and in consequence of the mediastinitis due to it, also after suppuration of the peribronchial lymph glands, etc. Of the acute infectious diseases it is especially hemorrhagic septicemia, pyosepticemia neonatorum, less often strangles, variola, articular rheumatism, etc., which lead to pericarditis.

It is only exceptionally that chemical substances or mechanical irritants give rise to chronic pericarditis, the former in bacterial diseases of other organs, in nephritis, etc., the latter by new-growths, parasites, sterile foreign bodies. (Borchardt saw serous-hemorrhagic pericarditis with nephritis and hepatitis in two horses which suffered from lysol poisoning.)

In infectious non-traumatic pericarditis certain predisposing causes like cold, over-exertion, railroad transportation, etc., are of importance although they are not capable, of themselves, of giving rise to an inflammation.

Pathogenesis. The injury of the tissue of the pericardium and its endothelial lining by infectious or other noxious substances causes a desquamation of the endothelia, and the passing out of fluid constituents of the blood, and polymorphous leucocytes. Since in acute inflammations such fluid is rich in protein, a separation of the fibrin soon takes place. The coutiuuously escaping fluid exudate is not infrequently again absorbed by the less affected parts of the pericardial layers, whereupon the fibrin which in the meantime has been separated, together with the cellular constituents, remains in the pericardial sac. Frequently, however, the quantity of the exuded fluid exceeds that of the reabsorbed exudates, resulting in a continuously increasing collection of fluid exudate. On the contrary, less virulent bacteria, especially tubercle bacilli, also chemical and mechanical irritants, induce the passing out of a fluid which contains relatively little albumen, and principally or exclusively migratory lymphocytes, whereupon the separation of fibrin is insignificant, or entirely absent. In such cases, however, a proliferation of the connective tissne is stimulated, which of course may also become associated with the inflammations described, if they are of longer duration.

In acute, inflammatory conditions a reflex acceleration of the heart action results, by transmission of the sympathetic nerve of the heart, possibly also by local increase of the irritability of the heart muscle and its nerve ganglia. At the same time, the inflammation, as an extra stimulation of the irritable parts of the heart, induces very frequently an extrasystolic arhythmia of the heart. On the other hand, the friction of the hypersensitive and rough pericardial layers causes severe pain by the activity of the heart. The interference with the diastolic dilatation of the heart cavities, with a simultaneous compression of the vena cava by the fluid exudate, and at times by the large amount of fibrinous masses, is, however, of the greatest importance. In this manner the venous system becomes overcharged with blood; the arterial system, on the other hand, is insufficiently fed with blood, whereupon a diminution of arterial blood pressure with slowing of the blood circulation follows.

The influence on respiration is indicated by the interference of the blood flow from the pulmonary veins, on the one hand, and by retraction or even compression of the lungs on the other hand, in those cases of significant increase in size of the pericardial sac.

In infectious inflammations of a moderately slow course, continuous or periodical absorption of infectious substances takes place, which, however, in most instances are immediately dissolved, their endotoxins giving rise to fever and toxic proteins. Some, however, may escape the dissolution, inducing inflammations of other organs.

Anatomical Changes. In **acute pericarditis** delicate or thick fibrinous membranes are deposited on one or both layers of the pericardium which may, in larger animals, attain a thickness of 2-3 cm., and a fluid exudate accumulates in varying amounts in the pericardial cavity, which may be clear or very cloudy; it may contain floccules of fibrin and in traumatic pericarditis it has usually a foul odor. If fibrin is secreted copiously, the surface of the heart becomes uneven, villous (villous heart, cor villosum), and the fibrin masses may divide the pericardial cavity into irregular compartments (which are especially well marked in cattle). In many cases the exudation assumes a purulent character and if the inflammation is prolonged a proliferation of connective tissue is more manifest. The latter form of inflammation, and also pericarditis in its initial stage, is sometimes limited to portions of the pericardium (pericarditis circumscripta) in which the vicinity of the large vessels is affected preferably, less often that of the apex. In the majority of cases, however, the inflammation involves the entire pericardium almost uniformly (P. diffusa), although also in these cases the visceral layer usually shows more intense changes. In proportion to the amount of exudate the pericardium is more or less distended and sometimes occupies a

very considerable portion of the thoracic cavity. The quantity of exudate varies within wide limits and may amount to 36 (Friedberger) and even 40 liters in horses, 18½ liters (Trasbot) in cattle, and ½ liter in dogs. The connective tissue septa of the heart muscle are also infiltrated with sero-cellular exudate; if the amount of exudate is very great, compression and atrophy of the heart may result.

Finally there are evidences of stasis, such as marked fullness of the veins, passive hyperemia of the lungs and of the liver, edematous infiltration, etc.

In **traumatic pericarditis** of cattle the foreign bodies are not infrequently found in the exudate, either free or with one

Fig. 741. Initial stage of traumatic pericarditis with puncture of the left wall of the ventricle by a sewing needle. Funnel-shaped puncture in the heart muscle and a corresponding opening in the pericardial sac with a roughened character of the serosa surrounding the opening. The pericardium is here shown drawn back.

end penetrating the wall of the heart. It may also occur that the foreign body is not found because it has been pushed back into the reticulum, or because it had rusted and crumbled; in many cases it is entirely in the heart muscle, or exceptionally, in a ventricle. (It is possible for an abscess to form in the heart muscle and break into the pericardial cavity or into one of the ventricles [Cadéac].) A traumatic origin of the inflammation is suggested by the presence of a firm connective tissue cord between pericardium and diaphragm, and also between the latter and the reticulum. This cord is either solid, or it contains several cavities, either side by side or communicating with each other, and filled with purulent or ichorous fluid; in rare cases a long canal is found in the center of the connective tissue cord, which connects the lumen of the reticulum with the pericardial cavity.

Chronic pericarditis is characterized by connective tissue

proliferation leading either to simple thickening of the pericardial layers (tendinous spots, Maculae albidae s. lacteae) or to adhesions between them (P. fibrosa adhaesiva); sometimes, indeed, a complete union of the pericardial layers results (Concretio pericardii). In tuberculous pericarditis cheesy foci may be found in varying numbers in the new connective tissue which may be very copious, but this form of pericarditis occurs, especially in dogs, also as a serous or hemorrhagic inflammation.

Symptoms. The clinical picture of traumatic pericarditis is usually preceded by prolonged digestive disturbances, which, according to Bongartz, last from 1 to 6 weeks; sometimes they become very severe and may be associated on motion with sensitiveness to pressure in the region of the reticulum, with superficial respiration and evidences of pain. Later, when the foreign body has reached the pericardium, the symptom complex of pericarditis follows immediately; although exceptions may be noted in so far as the signs of traumatic gastritis may disappear for a time the pericarditis becoming manifest only after

Fig. 142. Lateral deflection of the elbow in traumatic pericarditis.

their reappearance, or the trouble may develop without previous digestive disturbances. In similar manner a non-traumatic pericarditis of secondary origin is introduced by the symptoms of the basic affection.

The period between the ingestion of the foreign body and the appearance of the pericardial symptoms depends upon the nature of the foreign body, the

manner in which the animals are kept and fed, and upon accidental factors (parturition, etc.).

In the **initial stage of pericarditis** the animals manifest severe pain, they avoid all motion, stand with head stretched forward, the elbows spread out, the hind feet drawn under the belly, the back arched, they frequently look back toward the abdomen and at times lie down carefully, only to get up again immediately. Pressure on the region of the heart, on the chest or on the back; also percussion of the precordia increases the pain considerably; the animals groan, attempt to evade the pressure or depress the back deeply. They dislike to move, are surprisingly fatigued after brief motion, and then show shallow breathing, respiration becoming abdominal in type. The pain increases markedly, especially on going down hill. These symptoms are sometimes so mild as to fail to arouse attention, and the first expert examination, especially in cows which are not worked, often shows already a fairly advanced stage of the disease.

The heart beat is hastened from the first by 1½, 2 or 3 times the normal rate and may be arhythmical. The increase is sometimes aggravated further without any cause whatever, or through quite insignificant influences. Especially during motion the heart beat shakes the entire region of the heart, and the heart sounds are sometimes heard at a distance of 3 to 4 steps, being unusually clear and loud. (Lecouturier describes them with the word "claque-claque"). Sonnenberg observed bradycardia in a cow (26 heart beats to the minute), *together with epileptoid convulsions and loss of consciousness*, which ceased during periodical occurrences of increased heart's action (over 100 heart beats).

Friction sounds in the heart regions afford the most valuable symptom. These sounds are synchronous with the motions of the heart, but they do not follow as regularly as the endocardial sounds; they may be heard together with the systole or with the diastole, or then immediately before or after. They may be soft or rough, creaking, and always give the impression of friction, or if they are very short and intense they sound as though two liquid or solid bodies were beating against each other. These sounds are often felt with the hand placed on the cardiac region, and may be increased on pressure upon the intercostal spaces. They are limited to the region of the heart, audible on both sides and are influenced by the respiratory movements at most by becoming less intense during inspiration, because then the lung has moved between heart and thorax.

In the presence of a fluid exudate, the friction sounds usually disappear entirely if delicate or few pseudo-membranes are formed separating the rough surfaces of the pericardium; in the presence of only small amounts of fluid the friction sounds may persist. If there are very copious masses of fibrin, some portions of the fibrin covered surfaces touch even in the pres-

ence of much fluid and produce friction sounds to the end.

In traumatic pericarditis gases and fluid are not infrequently present together, and then splashing, gurgling and similar sounds are heard in addition to the friction sounds, also the sound of falling drops, etc., etc.

The heart beat, which is at first increased in intensity, gradually becomes weaker and may at times be unnoticeable, but if much exudate is present, wave-like movements may be felt in the cardiac region. According to the observations of Jakob, upon alternate palpation of the region of the heart in a dog standing and in a dorsal position, the heart beats can be felt much more plainly when the animal is in the dorsal position, by reason of the gravitation of the exudate toward the base of the heart. With the weakening of the heart beats the intensity of the heart sounds diminishes and sometimes they disappear entirely.

Fig. 143. **Increased cardiac dullness in pericarditis with fluid exudate.** The dotted curved line shows the margin of the normal, the solid curved line the margin of the increased dullness. The straight dotted line indicates the middle line of the sternum, the figures indicate the ribs.

An increased cardiac dullness may be ascertained only in the presence of much fluid, of very much fibrinous exudate or of tuberculous new-formations. The increase then gradually occurs upwards, forwards and backwards; upward it may reach as high as the shoulder line and may, in small animals, reach forward to the second intercostal space, backwards in all animals to the 6th or 7th intercostal spaces (Fig. 143). In the lateral position the upper border moves slightly down, because then the heart lies somewhat further away from the thorax. In ruminants an absolute cardiac dullness is at first noticeable, which later enlarges in the manner indicated. Any dullness on the right side is always less marked than on the left side. In some cases of traumatic pericarditis in which the gases of putrefaction form or in which gases from the reticulum have reached the pericardial cavity, a tympanitic and even a metallic percussion sound of varying extent may be elicited on the upper border of the dullness.

The pulse is accelerated from the beginning; at first fairly full and tense, often arhythmical, later it becomes very small, thread-like, owing to compression and possibly to involvement of the heart, and the arhythmia usually increases.

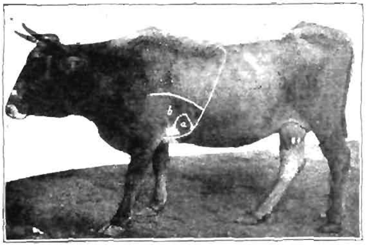

Fig. 144. Traumatic pericarditis with increased cardiac dullness and edema of the dewlap (before the aspiration of two liters of purulent exudate). a, Dull tympanitic sound; b, dullness. (Same cow as in Figs. 145 and 147.)

With the increase of the exudate the veins are constantly filled more strongly, and this is particularly noticeable in the jugular veins. The jugular veins, which are dilated like thick cords and stand out strongly, sometimes show undulations which are synchronous with the auricular systole and slightly precede the heart beat (presystolic venous pulse). However Zieger quite correctly calls attention to the fact that in the beginning the turgidity of the jugular vein can be detected only by a palpable stiffness of the wall of the vein, while exceptionally a swelling of the veins and edema of the dewlap may be entirely absent, in spite of the collection of a large amount of fluid in the pericardial sac and extensive dullness (Rossi, Marek).

At the same time an edematous swelling develops on the upper and lower chest and also in the anterior portion of the lower abdomen, further in the region behind the elbow, on the dewlap and in the trachea where it may appear first, if the head is held low, and may be associated with edema of the larynx.

According to Zieger, the edema of the dewlap begins in the form of a wheal at the lowest point of the dewlap or under the jaw, and may temporarily disappear again or coalesce with similar wheals into an extensive edema. Such a wheal can be recognized only by carefully palpating the entire length of the edge of the dewlap, or, if necessary, by comparison with similar locations on a healthy animal of the same age.

Disturbances in respiration exist in all cases, the breathing being more or less hastened and superficial, and labored on the slightest motion. From time to time a feeble and very painful cough is heard. With the increase of the edema fluid accumulates also in the body cavities and interferes still more with respiration, which is now labored even when the animals are resting, the more so as the symptoms of stasis constantly increase and may give rise to pneumonia.

The temperature is either raised from the beginning or a rise occurs in the later course of the disease; it is not usually high, unless the underlying disease causes a considerable degree *of fever. When the temperature rises within the body,* chills and trembling are frequently observed and the temperature is unequally distributed over the surface of the body. In cattle traumatic pericarditis may in exceptional cases run an afebrile course.

With the development of the symptoms the general condition of the animals becomes constantly worse. Large animals avoid every movement as much as possible, and stand *with neck* stretched out straight, they lie down only rarely, very carefully and only for short periods; their eyes have a plaintive expression; smaller animals usually lie constantly on the ground. The desire to eat diminishes more and more, so that the patients become emaciated.

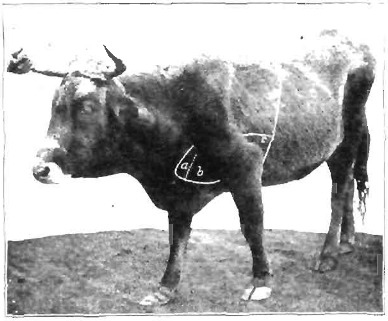

Fig. 145. Traumatic pericarditis with area of dullness. a, b, Anterior to the shoulder joint region; a, dull atympanitic sound; b, dullness; c, dullness posterior to the shoulder. (Same cow as in Figs. 144 and 147.)

Chronic pericarditis usually arises out of the acute form, the condition of the animals improving after a certain time, but without complete recovery. It is more rare for the disease to develop insidiously from the commencement (mostly in the presence of tumors, tuberculous swellings, etc.), and in such *cases the symptoms of the initial stage, like irregular heart* action, rapid fatigue during work, may remain unnoticed for weeks.

The *most striking symptoms* are dyspnea and rapid fatigue on moving about and still more when working, then a decided increase and arhythmic character of the heart function. Enlargement of the *cardiac dullness* is either absent or examination gives the same result as in acute pericarditis with effusion. In the latter case the heart beat and heart sounds are weak.

Friction sounds are usually absent. Another symptom is found in the fullness of the jugular veins. In the later stages of the disease edema may develop on the lower chest and in the vicinity.

Course and Prognosis. Acute pericarditis usually ends fatally, within the first week, in those cases in which purulent or ichorous exudate has collected in large quantities or in which the affection has occurred in connection with an infectious disease, especially pneumonia, pleurisy or hemorrhagic septicemia. In the majority of cases, however, the disease takes a longer course, 2 to 3 weeks and more, and shows at times transitory improvement. Ultimately, however, the permanent disturbance of the heart function or the degeneration of the heart muscle

Fig. 140. Intense filling of the jugular vein in traumatic pericarditis.

lead to death even in these cases the end being preceded by symptoms of stasis, such as venous hyperemia and edema, sometimes hemorrhagic infarcts in the lungs, chronic catarrh of the respiratory passages and of the intestinal tract, cutaneous edema, thoracic dropsy, ascites, etc. The animals become much emaciated and exhausted and usually perish in this stage with symptoms of pulmonary edema. Sometimes death occurs suddenly at a time when the general condition of the animal is still fairly satisfactory. Recoveries are rare in cases which are clinically well marked and usually follow upon a period of convalescence lasting several weeks. However, even in those cases which take a comparatively favorable course, chronic changes remain which influence the heart action unfavorably.

In extensive adhesions the heart beat is weak and the thorax is depressed in the region of the heart (Röll).

The course of traumatic pericarditis varies in the several cases, but is usually insidious, extending over weeks and months, and even longer, during which time the symptoms usually increase gradually or with transitory remissions. Eventually digestive disturbances become more and more evident and finally a soporose condition develops, accompanied by paralysis-like debility, probably due to the absorption of putrefactive poisons from the exudate (de Bruin). In many cases metastatic, purulent inflammatory foci form in various organs, for instance in the joints and tendon sheaths, and in such cases the disease may terminate with the clinical picture of pyemia. Other complications are pneumonia and pleurisy, gastric and duodenal catarrh, etc.

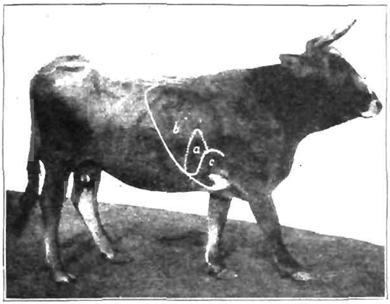

Fig. 147. Traumatic pericarditis with area of dullness on the right side. a, Tympanitic sound; b, field of percussion over lungs; c, dullness. (Same cow as in Figs 144 and 145.)

(In a case reported by Zieger, foul-smelling pus mixed with stomach contents was discharged through the nose of a cow while feeding. As a result of the penetration of the foreign body the right lung was almost entirely converted into a pus sac communicating with the reticulum, and this finally burst into a bronchus.) Cases of sudden death with well-developed clinical symptoms sometimes also immediately follow febrile traumatic disturbances of digestion of short duration or follow the edema of the throat and dewlap with but little perceptible acceleration of the heart (Metz, Becker).

Sudden death is not a very rare occurrence in any stage of the disease and follows either upon injury or perforation by the foreign body of the heart muscle or of the larger blood vessels of the heart, or through the influence of sapremia. On the other hand, protracted cases are observed occasionally, in which the well-marked symptoms gradually diminish in intensity, the nutrition improves and the animals may even be put to work (Mathis, Jensen). Sometimes spontaneous recovery occurs, the foreign body either breaking through to the outside or returning into the reticulum.

(Moussu recognizes such cures only in cases where the foreign body lies outside of the pericardial sac and produces only a nonpurulent inflammation of the same.)

Diagnosis. During the initial stage the symptoms are not sufficiently characteristic to make a positive recognition of pericarditis possible, although the development of an acute pericarditis is suggested by the very great sensitiveness of the precordia, and by the pounding, often arhythmic heart beat which is altered decidedly by even comparatively slight external influences. Later the friction sounds or the various splashing sounds, together with the decided enlargement and peculiar form of the cardiac dullness unite to make up a characteristic clinical picture, which is developed further by symptoms of blood stasis and by edema, and which it is almost impossible to mistake. It is, however, not to be forgotten that none of these phenomena are pathognomic in themselves and that some of them may be absent, not only in traumatic pericarditis but also in other inflammations of the pericardium.

For the diagnosis of a commencing traumatic pericarditis the high pulse rate (100-110 a minute) with a normal or at most maximal normal temperature (Meyer, Eber) is suggestive. According to Eber the question may be decided even in those rare cases in which the temperature rises more or less during the initial stage, the increased pulse rate remaining unchanged in spite of the lowering of the temperature by the action of antifebrin in doses of 50-100 Gm. per day. According to Otto a peculiar muscular trembling in the vicinity of the elbows is of importance in this respect, which occurs especially after the animal has been standing for some time.

Fibrinous pericarditis might be mistaken for fibrinous pleurisy when this has given rise to the so-called pleuro-pericardial sounds. These friction sounds are purely pleuritic and are characterized by being heard, together with the heart sounds, only during either inspiration or expiration; moreover, they are heard synchronously with the respiratory movements during the heart-pause. Moreover, pleurisy is rarely limited to so small a space and therefore friction sounds, synchronous with the respiratory movements, are also heard more posteriorly. Still, it must not be forgotten that pleurisy and pericarditis may occasionally be present at the same time.—In rheumatism of the intercostal muscles the intercostal spaces are painful, but friction sounds are absent or at most dull muscular sounds are audible. If a fibrinous pleurisy should also be present, as is the case occasionally in the so-called pleurodynia of horses, the nature of the disease will be sufficiently cleared up by the history of the case. From the endocardial sounds in valvular disease the pericardial friction sounds are differentiated especially by the fact that they seem to develop immediately under the ear, are sometimes increased by pressure and become weaker on inspiration; finally by the fact that they are not quite synchronous with the heart movements.—At the very beginning general acute infectious diseases may have to be considered, in ruminants especially anthrax, but in such cases the

temperature is usually decisive, since in pericarditis the temperature rises are usually slight unless the disease has developed upon a septicemic basis.

Pericarditis with effusion might give rise to confusion with pneumonia in those cases in which only the lower sections of the lungs, in the neighborhood of the heart, have become condensed and may therefore simulate an increased cardiac dullness. In such cases the character of the heart beat and of the heart sounds affords information because in pneumonia they are not weakened at all or only slightly so, in contrast to pericarditis with effusion.—In cattle, lung plague may cause a clinical picture which is somewhat like that of traumatic pericarditis, but in most cases sufficient points of difference are present, for instance, the preceding digestive disturbances, the comparatively small dullness, the striking weakness of the heart-beat, the complete absence of bronchial respiratory sounds, the characteristic pericardial sounds and slight fever or its absence.—In peritonitis with effusion an enlarged area of cardiac dullness may be simulated by an encapsulated exudation in the vicinity of the heart and, in larger animals, especially in cattle, by free fluid exudation. If free exudation is present in the thoracic cavity, the heart beat and the heart sounds are not weakened or only slightly, especially in smaller animals and in the sitting position; moreover, the dullness is limited horizontally.—An encapsulated pleural exudate or a swelling in the vicinity of the heart may be eliminated in such cases in which the heart has been displaced (see compression of the heart). A dullness due to enlargement of the heart (hypertrophy, dilatation) may have a similar shape, but hardly ever extends so far forward, the heart sounds are heard distinctly, friction sounds and sensitiveness of the precordia are absent.

About the nature of the exudate external conditions may offer information in so far as pericarditis occurring after cold, in consequence of croupous pneumouia, or of primary pleurisy leads to the formation of a fibrinous or sero-fibrinous exudate, while metastatic pericarditis is usually purulent and accompanied by high fever. In traumatic pericarditis of cattle the exudate may be sero-fibrinous, purulent or ichorous. The assumption of an ichorous pericarditis is justified by the greater severity of the general symptoms, by the tympanitic and possibly metallic nature of the percussion sound and by the presence of splashing sonnds. Traumatic pericarditis is usually manifested by local and venous symptoms similar to those of the rather infrequent tuberculous pericarditis of cattle (Kläber, Liénaux, Wyssmann and others). In dogs, however, it usually runs a chronic course, in the form of a sero-hemorrhagic or, less frequently, as a fibrinous pericarditis, with emaciation and without special disturbance of the general condition.

In doubtful cases Gmeiner recommends a test puncture of the pericardium, on the left side within the area of dullness, if necessary also on the right side.

Chronic pericarditis is very difficult of recognition without an exact knowledge of the history, because most of the symptoms are observed also in chronic pleurisy and in chronic interstitial pneumonia. Only an absolutely exact determination of the shape of the dull area, the nature of the heart beat and the characteristic symptoms of stasis may be of assistance. Chronic myocarditis cannot be differentiated with certainty from chronic fibrinous pericarditis.

Treatment. If treatment is justified by the fact that the prognosis is not absolutely unfavorable, complete rest must above all be assured. Any disturbance in defecation is to be relieved by small doses of mild laxatives, and the diet must be regulated appropriately. Herbivora should receive good hay, if possible mixed with fresh green feed, also flour- or bran-slop; carnivora are fed with gruel or broths, also milk. In persistent inappetence artificial feeding may become desirable.

The inflammatory process may be alleviated by cold compresses. A sac filled with snow or ice and fastened with a wide belt, or a compress made of tow or cotton, is applied to the region of the heart and cold water poured over it from time to time; large animals may be put under the shower. In primary pericarditis preparations of salicylic acid may be tried in similar doses as in pneumonia or in somewhat smaller ones, because their harmful action upon the heart is here more easily exerted. Lactophenin, salicylic acid, antifebrin, antipyrin and salipyrin may be administered in very high fever.

For weakness of the heart, cardiac remedies are indicated, digitalis (in large animals 2-4 gm. daily, in small animals, of a 2:150.0 infusion to which 5 gm. of potassium nitrate have been added, one tablespoonful every two hours). Caffeine is also of service (2-5.0 gm. or 0.2-1.0 gm. subcutaneously), camphor (camphorated oil 20-30 gm. or 2-3 gm. subcutaneously), possibly wine, alcohol, or ether.

To promote absorption diuretic remedies and mild laxatives may be tried. In excessive accumulation of fluid, with consequent compression of the heart, puncture of the pericardium should be considered. But in these cases puncture is not as harmless a proceeding as in pleurisy because a portion of the exudate may flow into the pleural cavity through the opening in the pericardium, after the trocar has been removed (Moussu, Gmeiner, author's case).

Puncture of the pericardium is done with a simple trocar immediately above the parasternal line, in the 5th or 6th intercostal space, but always within the area of dullness; after removal of the exudate the pericardial cavity may be irrigated with lukewarm sterilized water or with a mild disinfecting solution. If the exudate is purulent it seems more advisable to open the pericardium by an incision and to wash out the cavity thoroughly. During this operation it may be possible to find and remove the foreign body (Bastin).

In traumatic pericarditis Zieger had good results in three cases following complete removal of all fluid from the organ by

tapping in the intracostal space. In two cases, however, the animal had to be slaughtered, because a nail located in the pericardial sac wrought new injury to the heart muscle with each heart beat, which in turn was accompanied by severe hemorrhage. Under certain circumstances Meyer's operation may also be tried (see page 122). If this is not followed by improvement or if the disease is already far progressed slaughtering of the patients should not be delayed, because later on the flesh will become useless for human consumption on account of emaciation or sapremia or of general pyemic infection. In order to remove edematous infiltration and thus make the flesh fit for consumption Moussu recommends the removal of the exudate through a puncture from the direction of the xiphoid cartilage (Fig. 148). This procedure merits consideration also in non-traumatic pericarditis of cattle.

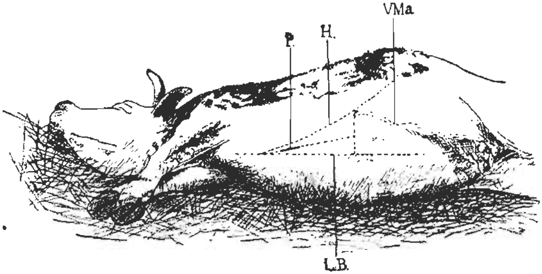

Fig. 148. Field of operation for puncture of the pericardium from the xiphoid cartilage. LB, Linea alba; H, costal arch; VMa, milkvein; P, point of incision. (After Moussu.)

With the animal standing, the positions of the xiphoid cartilage, the linea alba and the left costal arch are ascertained. In the middle of the angle outlined by these landmarks, above 20 cm. in front of the place where the milk vein runs toward the V. epigastrica cranialis, the abdominal wall is pierced, an incision is made through the skin about 20 cm. long, parallel to the costal arch, through which a portion of the serous fluid which has accumulated in the subcutaneous connective tissue will escape promptly. The muscles in the bottom of the wound are then separated with the scalpel as far as the neck of the xiphoid cartilage, the subpericardial fatty connective tissue is forced apart with one or two fingers of the right hand until the pericardium is reached, as is shown by feeling the heart beats. The left index finger is now pushed up to the pericardium, and upon it a trocar, at least 25 cm. long and 5 mm. thick is carried forward with the right hand until the heart beat is communicated to it. The trocar is now pointed inward and forward and is forced with a sharp thrust about 3-4 cm. into the pericardium, the fluid being allowed to escape by removing the stylet. A strip of iodoform gauze is placed into the wound, a compressing dressing is applied to the chest and renewed from time to time. The edema disappears, in the course of a few days, for a time, when the animal may be slaughtered.

Liénaux calls attention to the fact that in the presence of adhesions the operation fails just at this point of punction, or that the trocar may enter the ventricle, in which case a stream of blood will escape through the trocar. This accident appears however to be of no importance.

In order to prevent possible complications (phlegmons, malignant edema), Liénaux drains the pericardium, after punction, by introducing a rubber drain with lateral openings through the canula into the pericardial sac and securing it with sutures after removal of the canula. In a case treated in this manner the exudate disappeared permanently so that the animal could be fattened.

Chronic pericarditis can hardly be treated successfully. At best the animals must be saved from exertion and receive cardiac remedies. Animals which are intended for slaughter should be killed as early as possible.

Compression of the heart (so-called pseudopericarditis). Aside from the compression of the heart which is produced by a copious pericardial exudate, the organ may be crowded or displaced by formations outside of the pericardium; this occurs especially in cattle, but sometimes also in other domestic animals. Abscesses (mostly traumatic in origin) develop with preference subpleurally or subpericardially, and new-formations or the somewhat more frequent tuberculous swellings are found at the base of the heart or in the anterior mediastinum. Exceptionally the greatly enlarged thymus gland may exert pressure on the heart, lungs, and esophagus (Naudin). At times the compression is caused by a prolapse of the abdominal organs, especially the rumen, further also possibly by a hernia diaphragmatico-pericardialis, which occasionally occurs in dogs (Antonini, Forgeot, Stoeber, Joest). Sometimes the heart may also be compressed by marked tuberculous infiltration of the lungs (Marek), by abscess in the lungs or by very large actinomycotic areas in the lungs. If such formations are present, the diastolic dilation of the heart is more or less interfered with and at the same time the heart muscle is made to atrophy (Atrophia cordis). The large vascular trunks are often compressed at the same time, or they may be affected exclusively.

The clinical picture shows great similarity with that of pericarditis; especially similar symptoms of stasis are to be noted. On the other hand, the pulse rate is only slightly increased, intermissions are frequent and a displacement of the heart to the right, forward, backward or upward may be demonstrated by the character of the heart beat or of the heart sounds; pericardial sounds are absent. In certain cases the dullness is much larger than is usual in pericarditis. Sometimes an exploratory puncture affords valuable indications for diagnosis. J. Schmidt observed in a horse with sarcomatosis of the epi- and pericardium a marked dyspnoea, with sounds simulating those occurring in roaring, even after short exercise. In Munich's case in a cow the prolapse of the rumen into the thoracic cavity induced cough, weakening of the respiratory sounds following feeding, and later sudden death without previous signs of stasis.

Treatment need be considered only in the case of subpleural or subpericardial abscesses or of cystic new-formations, in order to remove the fluid contents.

2. Dropsy of the Pericardium. Hydrops Pericardii.

(*Hydropericardium.*)

Etiology. With the exception of inflammatory processes,

which do not belong here, an accumulation of serous fluid occurs in the pericardium either from stasis of the blood in chronic diseases of the heart muscle or of the valves, or in chronic pulmonary affections, and also in connection with hydremia. On a similar foundation dropsy of the pericardium develops in chronic inflammations of the kidneys, in chronic glanders, chronic anemia, in liver-fluke disease, lungworm disease, etc. The diseases which have been enumerated usually exert their effects also upon the veins of other parts of the body, and therefore symptoms of hydrops are in these cases observed elsewhere also.

Anatomical Changes. The pericardium of healthy animals contains at most a few cubic centimeters of a serous fluid, but in dropsy of the pericardium the quantity may amount to 100-200 times the normal. The liquid is clear or slightly turbid (because of the admixture of desquamated and degenerated epithelial cells), it is thin fluid, sometimes bloody; the specific gravity is less than 1016, the content of albumen less than 3%. Other changes are present which point to the underlying condition.

Symptoms. Dropsy of the pericardium is always observed in animals which have presented symptoms of the basic disease for some time. Aside from these the most important symptoms are an enlarged area of cardiac dullness of similar shape and extension as in pericarditis with effusion, feeble and dull heart sounds. If dropsy is present also in the thorax, the enlarged area of cardiac dullness is in small animals easily determined while they are sitting or lying on the back, because then only the dullness due to the fluid accumnlated in the pericardial cavity remains.

Symptoms of stasis, respiratory disturbances, albuminuria, feeble and small pulse beats also become evident, but these symptoms are largely due to the basic condition itself which indeed is of deciding influence upon the course and prognosis.

Diagnosis. Dropsy of the pericardium differs from pericarditis with fluid exndate by the absence of acnte symptoms (pain, fever) and of friction sounds, and by the presence of manifestations of hydrops elsewhere.

Treatment. As hydrops of the pericardinm only represents a phenomenon of other affections, the treatment must be adapted to the nature of the underlying condition. It is sometimes possible to diminish the pericardial fluid by the systematic administration of diuretic and diaphoretic remedies and of laxatives. If the accumulation of fluid is excessive puncture of the pericardium may be attempted.

Accumulation of Blood in the Pericardium (Haemopericardium). Hemorrhage into the pericardial cavity may occur from the cavities of

the heart, from the blood vessels of the heart muscle or from the larger vessel trunks. As immediate causes of bleeding may be mentioned trauma or spontaneous rupture of the heart, rupture of the dilated coronary artery or of a large vessel trunk, especially of the aorta, if this is dilated above the valves. Finally bleeding may occur exceptionally in diseases with hemorrhagic diathesis.

Hemopericardium can only be recognized with a certain degree of probability in cases in which the bleeding is slow, because in rapid hemorrhage death occurs as soon as in ½-1 hour. The diagnosis is based upon the demonstration of fluid in the pericardium (p. 745), with symptoms of internal hemorrhage.

3. Gas in the Pericardium. Pneumopericardium.

Etiology. An accumulation of gas in the pericardium occurs usually only in cattle, when a foreign body penetrating into the pericardium from the reticulum has formed a canal through which gases may pass from the reticulum into the pericardial cavity (see p. 744); the same occurs in ichorous pericarditis in which putrefactive gases are formed. Atmospheric air enters the pericardium only in very exceptional cases, for instance after penetrating wounds of the pericardium by external agencies, by fractured ribs, by breaking down of a lung cavity which had been adherent to the pericardium.

Symptoms. An intense tympanitic percussion sound is noticeable in the region of the heart, over an area which is sharply limited at the periphery and corresponds to the cardiac area; this sound is sometimes metallic in character. In pure pneumopericardium the heart sounds are high and metallic, owing to the resonance of the pericardium. In most cases, however, the pericardium contains fluid exudate as well as gases, and this gives rise to loud splashing, clacking noises which are sometimes audible from a distance of several steps, and which are often compared with the noise made by a millwheel (bruit de moulin).

The clinical picture is supplemented by the more or less marked symptoms of pericarditis (p. 744) which is usually present, or the signs of gas accumulation appear only in a certain stage, for instance, at the beginning or toward the end of pericarditis. In making a diagnosis it should be remembered that a tympanitic sound in the region of the heart may also be produced by infiltration of the lung portions in the vicinity of the heart or by diaphragmatic hernia.

Prognosis. Pneumocardium is always of very unfavorable significance and a chance for recovery exists only in those rare cases of injury to the pericardium in which infection failed to occur.

Treatment. The same treatment is here indicated as in acute pericarditis but the necessity for operation is greater

SECTION II.

DISEASES OF THE HEART.

1. Paroxysmal Acceleration of the Heart. Tachycardia paroxysmalis.

(Palpitation of the heart; Palpitatio cordis.)

Paroxysmal tachycardia designates a suddenly occurring attack of pronounced acceleration of the heart, which suddenly disappears after minutes, hours or days, developing in the course of organic heart diseases (secondary paroxysmal tachycardia) or without organic changes in the heart and its blood vessels (essential paroxysmal tachycardia).

The term palpitation of the heart is less suited for the designation of such attacks, since human physicians understand by this term only the peculiar feeling in the region of the heart which appears, with or without objective signs, in the form of an increased and accelerated activity of the heart in all varieties of diseases of the heart.

Etiology. Exceptionally paroxysmal tachycardia may develop as a result of nervous influences, especially in highly bred and temperamental horses and dogs, due to increased direct or reflex irritation of the sympathetic nerve, or under certain condition of the automatic heart ganglia or of the heart muscle itself. The first mode enters into consideration in those cases in which palpitation occurs after violent psychic irritation, after fright, in case of fire, on falling, after railroad transportation, or from over-exertion; similar conditions may, however, also lead to acute cardiac dilatation or to acute heart weakness. A congenital morbid irritability of the nervous system in nutritive disturbances, in hemorrhages, in Basedow's disease, must in certain cases also be brought into causal relation with palpitation. Then inflammation or degeneration of the N. vagus (in chronic lead poisoning) usually leads to tachycardia. Finally palpitation sometimes persists after influenza (Pr. Mil. Vb.).

Rothberger & Winterberg and Haring produced by faradic and toxic stimulation (adrenalin, nicotin) of the acceleratory nerves, excessive, ventricular extra systole, and thereby tachy-

cardia attacks, whereas purely nervous stimulations proved effective only in special predispositions.

In animals the paroxysmal tachycardia originates probably in most cases from various organic heart affections, associated with acute or chronic weakness of the heart, in the course of which there is ample occasion for the appearance of excessive extra systoles.

Symptoms. The entire behavior of the animals, especially their distressed expression, reveals great anxiety, the animals tremble, horses and cattle perspire. In the heart region thrills are seen which are synchronous with the heart beat and which are sometimes transmitted to the entire trunk and to the vertebral column. The heart beat is stronger, as a rule much accelerated and arhythmical in so far as a number of rapid heart beats alternate with several which are less rapid, or as brief intermissions occur. The heart sounds are very strong and are sometimes audible from a distance of several steps. At the same time the pulse is usually feeble. These symptoms permit the assumption that a heart weakness was present in the cases in question, and this assumption is strengthened by other signs mentioned by several authors, such as intermittent pulse, systolic murmurs, greater fullness and pulsation of the jugular vein.

Diagnosis. This is based on the occurrence of pronounced acceleration of the heart, and the disappearance of all disturbances after such attacks. This distinguishes the paroxysmal tachycardia from the prolonged or lasting acceleration of the heart in the course of organic heart affections, acute infectious diseases, and in the most varied internal affections. Spasms of the diaphragm, as well as the chronic spasms of the abdominal muscles, distinguish themselves by the absence of tachycardia, and by the presence of twitchings in the region of the costal arch and abdominal walls.

Course. The attack usually disappears rapidly or gradually after hours, and at the longest after days; it may, however, recur from outside influences, or in anemic and otherwise predisposed animals.

Treatment. An attack of true tachycardia usually passes over without any treatment after complete rest is secured and the diet regulated. In nervous excitement the following drugs may be administered: morphine (for horses 0.30-0.50 gm., for dogs 0.02-0.05 gm. subcutaneously), chloral hydrate (20-40.0 or 1-2.0 gm. dissolved in lukewarm water, per os or per anum), possibly potassium bromide (10-15.0 gm. or 1-2.0 gm. in the drinking water).

Turan achieved a decrease in the number of heart beats in man for a period of days or weeks by intravenous injection of pure calcium chloratum crystallisatum (5 cc. of a 20% solution).

Cardiotonic remedies should be administered if cardiac weakness is present.

2. Slow Heart Action. Bradycardia.

Slowing of the heart action depends upon a functional disturbance in the heart muscle without any demonstrable organic changes.

Etiology. The cause of a decided slowing of the heart beat lies either *in an excessive stimulation of the vagus or* in certain affections of the heart muscle.

An excessive stimulation of the N. vagus may follow upon a bilateral compression of this nerve (in the thorax cavity, in the medulla oblongata), it may be due to slight inflammation, to compression of the vagus center in certain brain affections, and finally, it may be a reflex effect from the stomach or intestine, from the peritoneum or from the skin (Albrecht). Certain chemical substances (e. g., tannates) are also capable of irritating the vagus. Certain diseases of the heart muscle frequently cause bradycardia, for instance, degeneration of the muscle in acute infectious diseases, chronic myocarditis, atrophy of the heart, affections of the coronary arteries. In such cases the decrease in the number of heart beats may be brought about by the regular omission of every second contraction of the heart or by dissociation of the heart (see Adams-Stokes syndrome). On the other hand, insufficient nutrition of the heart muscle in cases of advanced inanition leads to bradycardia by retarded stimulation at the points where the veins enter the heart.

Symptoms. In horses a reduction in the number of heart beats has been observed to as few as 9 (Zimmermann), in most cases only to 12-20 (Dieckerhoff, Nordheim, Vogel, Zschokke), in dogs to 20-18 (Fröhner, Albrecht). The heart beat remains vigorous and the heart sounds are clear. The animals are apparently quite well; only in severe cases they show, aside from the symptoms of the underlying disease, languor, somnolence, lassitude, attacks of heart spasms or of heart weakness.

Diagnosis. Bradycardia is easily recognized and the cause may often be ascertained. It is possible to determine by subcutaneous injections of atropine (for large animals 0.02-0.05; for dogs 0.005-0.008 gm.) whether the cause lies in the vagus or in the heart, because bradycardia which is due to an excessive stimnlation of the vagus disappears for a time after the administration of atropine. Symptoms of heart weakness, which may become manifest only on motion, point to an organic disease of the heart, similarly as does the inability to influence bradycardia by atropin or at most a retarded and slight acceleration of the pulse following an injection of atropin.

Treatment. Nerve stimulants are indicated in all cases, such as injections of atropine, alcohol, ether, also sponging, and the relief of any digestive disturbances that may be present.

3. Intermittent Heart Action. Actio Cordis Intermittens.

(*Arhythmia cordis.*)

Intermittent heart action is a disturbance of the heart beats which may be produced by the total omission of one or several ventricular contractions or by additional systolic contractions.

Etiology. The omission of ventricular contractions occurs frequently in horses as a sequel of acute infections diseases especially in the course, or during the convalescence, of influenza; in other animals it is hardly ever observed. As the actual cause a reflex or immediate excessive stimulation of the N. vagus may be assumed, since in the cases observed by the author the omissions could always be relieved by atropine, which is known to paralyze the vagus endings. The stimulation of the vagus becomes manifest by the intermediation of the central nervous system when the omissions occur in brain affections, for instance, in case of increased intracranial pressure (chronic hydrocephalus internus, brain tumors, inflammation of the brain coverings, etc.). Digestive disturbances (enteric catarrh, obstipation) also sometimes give rise to transitory intermissions which are probably produced by chemical substances that have been absorbed from the intestine, in such a manner that the poisons irritate the N. vagus or its center either directly or reflexly. The ventricular systole may finally be omitted in certain cases in connectiou with diseases of the heart muscle or of its nervous apparatus, for instance, in acute inflammatory processes in the heart muscle, in the pericardium or in the endocardium.

Intermissions of the heart beat which are produced by additional systolic contractions occur in all animal species and always depend upon an abnormal stimulation of the heart. The abnormal stimulation consists either in an increased diastolic pressure in any one portion of the heart (in heart weakness), or it develops in acute inflammatory processes in the heart muscle, in the pericardium or endocardium. Sometimes poisons which have been absorbed in the intestines exert an abnormal stimulation upon the heart.

Pathogenesis. An excessive stimulation of the vagus reduces the physiological irritability and the conductivity of the heart for stimuli, this occurring first at the point of passage between the auricles and ventricles, where the stimuli from the auricles to the ventricles are transmitted only by the narrow His's bundle. Under such conditions His's bundle, because of its slight conductivity, responds less quickly to the stimulation coming from the atrium and also tires very soon as a

result of which the transmitted stimulation is increasingly delayed in its arrival at the point of contact at the origin of His's bundle (Tawara's or atrioventricular node) so that sooner or later it is no longer able to produce a contraction of the ventricle. During the following longer pause, however, it regains its original conductivity. In diminishing the conductivity in the wall of the auricles a missing of the auricular contraction may also ocur. Similar results may follow affections confined to the His's bundle, or to diffuse affections of the heart muscles (degeneration, inflammation, induration, neoplasm).

Abnormal stimulation acting upon the heart at the time of diastole produces an additional systole, thus prematurely interrupting the diastole. The additional systole of the ventricle follows the preceding normal systole more or less prematurely, but by reducing the irritability and conductivity of the heart muscle for a short time prevents the occurrence of the next following physiological systole (ineffectiveness of the sinus stimulation), and in this manner permits a longer or shorter pause (compensatory pause) to occur the sooner it has followed the preceding normal systole. The additional systole produces

Fig. 149. Intermittent heart action in the horse. Spygmogram of the abdominal aorta. Pause between successive pulse beats (time indicated in 0.2").

no perceptible arterial pulse, since immediately following the preceding normal systole the fill as well as the contractibility of the ventricle is still too small to force an appreciable quantity of blood into the aorta, in which the blood pressure has but slightly decreased at this time, and hence prevents the emptying of only the weakly contracting left ventricle. In case of an additional systole, atrium and ventricle either contract simultaneously or in too rapid succession, and by destroying the already partly completed stimulation of the sinus produce an omission of the next beat of the heart. On the other hand, a seldom occurring additional systole of the atrium also produces two heart beats in rapid succession; however, the pause following the additional systole is but little longer than the pause between two normal contractions of the heart.

Symptoms. In **omission of the ventricular systole** a pause corresponding to one heart beat can often be observed after every 2 to 5 heart beats. The intermissions are, however, often much less frequent, only after every 10 to 15 heart beats, and occasionally no regularity can be observed. Usually only one heart beat is omitted, but there are cases in which 2 to 4 successive heart beats are missing (Fig. 149). In many cases a very feeble, dull, short heart sound is heard in the anterior portions of the heart, which corresponds to the persisting auricular contractions, while the vigorous systolic heart sound which depends upon a contraction of the ventricles is omitted. The occurrence of the auricular contraction may be assumed positively because the negative venous pulse at the entrance of the chest can be felt even during the pause (Fig. 150). The heart beat following upon the pause is usually stronger, while the subsequent beats are either normal in force, or evenly

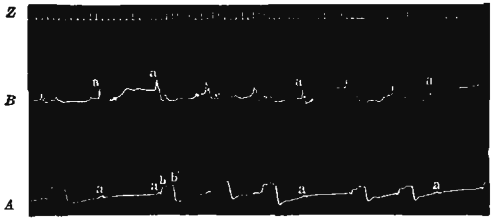

Fig. 150. **Intermittent heart action and contractions of the ventricles, occurring at different times, in a horse.** *A* Cardiogram, the small apices *a* correspond to the auricular contractions, the larger *bb'* to the ventricular contractions. The auricular apex persists during omission of ventricular systole. It corresponds with *a* of the phlebigram *B* of the jugular vein (negative venous pulse). After each omission of the ventricular contraction the pressure in the vein rose, because the vein was not emptied. (*Z* time in 0.2".)

weakened, or then gradually diminish in intensity. The pulse behaves similarly to the heart beat (true arhythmia of Caroni and Cadiot). During motion the number of heart beats is increased, the abnormal rhythm being usually preserved, although sometimes it changes in the same animal from time to time, so that the pauses occur at shorter, and again at longer intervals. Dreyer observed in a horse a *transitory disappearance* of the missed beat following an eserin-arecolin injection.

Extrasystolic arhythmia is recognized by the rapid succession of two or several heart beats, which are separated from the other, normal, heart beats by a pause, so that bigeminous, trigeminous, etc., heart beats are noted. The pulse beat corresponding with the additional systole is usually absent; only when this systole is separated from the preceding one by a

longer interval, or if it occurs frequently a rather feeble pulse *may be felt (Fig. 151). During motion the arhythmia* becomes still more striking, or if it had not been observed at rest it then becomes manifest. It may often be relieved temporarily by cardiotonics.

Course. If the intermission of the heart action occurs in the course of an acute disease, it disappears together with its removal; but if in the meanwhile lasting changes have developed in the heart, the arhythmia persists without interfering, as a rule, with the health of the animal. In chronic heart affections, however, the arhythmia disappears at most for a certain time and the animals show signs of being broken-winded.

Treatment. Aside from the treatment of the basic disease, animals which show a considerable degree of arhythmia should

Fig. 151.* *Arhythmia of the heart due to additional systole,* in a dog with cardiac dilatation and failure of the bicuspid valve. *c* Curve of heart beats. *A.f.* Sphygmogram of the Art. femoralis, *Z* time in 1/5 seconds; *s* normal ventricular systole, *the pulse (s) in the sphygmogram is distinctly felt; s'* additional systole, to which at + in the sphygmogram no beat corresponds, but at *s'* a minimal rise; *s'-s''* crowded additional systoles, to which elevations in sphygmogram correspond which are in part minimal and are hardly or not at all to be felt.

be worked only with great care. If the arhythmia depends upon organic diseases of the heart muscle, *cardiac remedies are sometimes of value.*

Adams-Stokes Syndrome. (Heart block, Dissociation of the heart.) By this term physicians understand a permanent and spasmodically increasing brachycardia with spells of disturbance of consciousness and paleness often accompanied by epileptiform convulsions. It is due to the independent functioning of the auricles and ventricles (dissociation), brought about by the absence of the transmission of the stimulus from the auricles to the ventricles. This occurs in cases of extensive lesions in His's bundle (in all cases in horses so far reported, by inflammatory infiltration), in which case the ventricles, by virtue of their automatic function, continue to contract at a slower rate than, and independently of, the auricles, which normally contract more rapidly. Under certain conditions the phenomena may be due to an inadequate response of the

muscles of the ventricle to otherwise unimpeded transmission of auricular stimuli in cases of extensive changes in the heart muscle.

In veterinary medicine, five cases of dissociation of the heart in horses have been described (Liénaux and Hermans, Charlier, O. Bang, G. and O. Petersen). Particularly in the three cases reported by Bang and G. and O. Petersen the presence of a heart block was ascertained by also taking the venous pulse. In spite of the usually more rapid contraction of the auricles, brachycardia continues (18 to 20 pulses per minute). The pulse rate following exercise will be changed only insignificantly, or the reduction in the pulse rate becomes more pronounced and the animal becomes breathless or collapses. Brachycardia may also occur in spells with intermittent functioning of the heart and pronounced venous pulse which is more rapid than the arterial pulse. Spells of loss of consciousness with collapse of the animal while at rest may also occur. Besides dilatation of the heart, Liénaux and Hermans observed pronounced brachycardia which even increased with exercise but was later followed by increased rapidity of heart action, as in fever. In Charlier's case pronounced brachycardia (15 to 16 heart beats per minute) was present and hardly changed even when the animal was exercised in a trot, while the animal showed spells of loss of consciousness and collapse. In all cases atropin has no effect upon brachycardia. The usefulness of the animal is greatly decreased or becomes entirely negligible.

In differential diagnosis simple brachycardia must be considered. In this ailment, however, in the horse and in the cow, the jugular pulse is easily palpable and often visible and exhibits the same decrease in frequency as the heart beats and the arterial pulse.

In the way of treatment, stimulating drugs (caffein, camphor) may be considered, also injections of eserin with atropin in medium doses (2 to 3 times daily), with which Semerau observed the disappearance of the symptoms in one case in man. In horses the condition has thus far proved incurable.

4. Heart Weakness. Cardiac insufficiency. Insufficientia cordis. By the term "heart weakness" is understood a decreased capacity of the heart muscle, either only under extraordinary strain (relative heart weakness) or under ordinary conditions of care and use (absolute heart weakness). In severe cases absolute heart weakness may appear under conditions of absolute rest (insufficiency while at rest), or in light cases symptoms may become apparent only during bodily exercise which does not exceed the physiological or average degree (insufficiency during exercise).

A separate presentation of heart weakness seems justified because of the fact that it frequently rests upon purely func-

tional disturbances of the heart muscle, and that it may occur in the course of various diseases of the heart without the possibility of determining the anatomical basis in all cases during life, and finally that it often requires special treatment without regard to the basic changes.

Etiology. Relative insufficiency occurs in animals quite frequently in the course of a few minutes or hours in the form of acute heart weakness as a result of bodily overexertion, especially at a time of unaccustomed muscular exertion. Under such conditions the diastolic fill of the heart cavities is very much increased by the excess venous inflow, while at the same time the arterial resistance is the more increased by the violent muscular contraction, since frequently during unaccustomed muscular exertions irrational and insufficiently modified impulses of exertion are carried to the muscles and the rational nervous stimulation of the blood vessels is probably absent. These influences are especially detrimental if the heart muscle has previously been subjected to only slight exertions through periods of rest lasting for weeks and months and has also decreased in size (atrophy through inactivity). The same is true of weak animals and also of well-conditioned fat animals, which are endangered by the excessive body weight as compared to the size of the heart muscle. This form of acute heart weakness is most frequently observed in army horses and race horses and in these animals is called "heart fatigue" or "acute insufficiency of the heart." Such cases were frequently observed by Lorscheid during the World War in army horses, and he was able to trace them to great exertion with insufficient food or to chronic diseases of other organs (see also Vol. 3, page 809).

Acute heart weakness resting upon a similar basis not infrequently occurs also in cattle and hogs, if the animals, after long-continued stabling or in a high condition, especially when being driven, are forced to undergo unaccustomed muscular exertion and thereby probably execute unnecessary movements by jumping around or as the result of stubbornness. Similar phenomena may also be observed in dogs which have become overheated while hunting.

Acute heart weakness in the form of an absolute insufficiency may also be observed in infectious diseases as well as in acute diseases of the heart muscle or other diseases of the heart resulting in parenchymatous and fatty degeneration or atrophy of the muscle fibers. It may also result from the detrimental influences of toxins.

Chronic heart weakness develops slowly, in the course of days, weeks, or months, and seldom occurs in animals under the influence of continued excessive work (race horses, heavy draft horses, draft dogs). As a rule it develops in the course of

insufficiency of the heart valves in diseases of the heart muscle (fatty heart, extensive parenchymatous degeneration, myocarditis, neoplasms, parasites), in organized adhesions of the pericardial sac, and in the course of diseases of the coronary arteries, which in animals, however, are seldom diseased. Heart weakness may also sometimes be traced to diseases of the kidneys, since by general contraction of the blood vessels on the one hand, and by probable excessive retention of water on the other hand, it may bring about a pronounced increase in the arterial resistance. Quite frequently chronic pulmonary emphysema can be found as the cause of heart weakness.

Pathogenesis. In the course of bodily overexertion even a healthy heart muscle may become more or less incompetent as a result of overdilatation because it can no longer propel the large inflow of venous blood against the extraordinarily increased arterial pressure. During the further increase in the resistance, the expelled mass of blood becomes smaller and smaller until in the severest cases the blood no longer leaves the heart cavities and the heart finally stops while engorged with blood. Under such conditions a poorly nourished heart muscle or one that has become weak as the result of long-continued rest is even more likely to break down. In case of an absolute decrease in the power of the heart a decrease in the volume of blood propelled takes place during complete bodily rest or during muscular exertions that do not exceed the average amount in daily life, because the weakened heart muscle can now no longer overcome the average arterial resistance, by reason of the decrease in its contractile force as a whole, or of the number of contractile muscle fibers. The diseased heart muscle no longer possesses the required tension to overcome the arterial resistance, but relaxes under the force of the blood flowing in during diastole.

In case of a weakness of both halves of the heart, the inevitable result is a venous stasis with increased venous blood pressure going hand in hand with the degree of weakness. The arterial pressure, on the other hand, may remain normal, because in light cases and in cases of medium severity a compensating tonic contraction of the smallest arteries takes place, because an increase in the carbonic acid contents and an increase in the acid products of metabolism develop during stasis of the venous blood. As a result of this, the blood pressure in the arteries lying toward the heart is kept on an average height in spite of a decrease in the volume of blood propelled. In case of excessive contraction of the blood vessels, the blood pressure may even be increased above normal (excessive stasis, according to Sahli). A decrease in the arterial pressure takes place only in case of severe heart weakness due to the now very much decreased power of propulsion. Under all conditions the increased venous pressure results in a de-

rease in the pressure difference between the arterial and venous system, with an accompanying decrease in the rapidity of the flow in the capillaries. The veins and capillaries now become engorged with blood, so that the decrease in the velocity finally results in an abnormal distribution of the blood throughout the vascular system. The decrease in the rapidity of flow may also be due to a great extent to the disappearance of the regulatory mechanism of the capillary walls. The walls of the capillaries are probably also directly damaged by the superabundance of carbon dioxide, lack of oxygen and accumulated products of metabolism.

An unequal decrease in weakness in the two halves of the heart could endure for only a few hours or less, for otherwise all the blood would be crowded into one side of the circulatory system within a comparatively short time. For a certain length of time such a condition, however, is nevertheless possible; it may, under certain conditions and in a slight degree, even endure permanently and present the phenomena of heart weakness predominantly affecting only one side of the heart. A weakness of the left side of the heart particularly results in an insufficient fill of the arteries of the body, accompanied by a decrease in the rapidity of flow in the capillaries. It furthermore results in engorgement of the veins in the lungs, with stasis in the lesser circulation, due to stasis of the blood in the left auricle. On the other hand, a weakness of the right side of the heart produces stasis of the blood in the veins of the body, poor circulation in the lesser circulation and in the arteries of the body.

The activity of the organs and also of the heart muscle must necessarily suffer from a decrease of rapidity of flow in the capillaries, since this decreases the supply of oxygen for the body cells, increases the carbon dioxide tension, and permits the accumulation of waste products. Furthermore, the activity of the heart is increased, this being brought about by the combined effects of an irritation of the center of acceleration (due to increased blood pressure), overdilatation of some portions of the heart (due to a stasis of the blood), and probably also accumulation of waste products. Because of the injurious effects of a decreased rapidity of flow, the walls of the capillaries become more permeable and less able to reabsorb the body fluids, as a result of which edematous swellings are readily formed in the course of pronounced or long-continued heart weakness.

Symptoms. Acute heart weakness may appear suddenly with symptoms of grave insufficiency of the circulatory system following severe and particularly unaccustomed overexertion. It manifests itself in severe dyspnea, cyanosis, and frequently edema of the lungs; furthermore, in pronounced acceleration

and extreme weakness of the pulse, engorgement of the veins, and profuse perspiration, whereupon the animal soon or suddenly collapses and dies in convulsion (paralysis of the heart). Such unexpected deaths are sometimes observed in race horses, hunting dogs, and especially in cattle and hogs while they are being driven. On the other hand, very mild cases of acute insufficiency of the circulatory system may be observed with light dyspnea, debility, rapid tiring, slight perspiration, increased pulse rate up to one and a half times to twice normal with a corresponding weakness of the pulse, and pounding of the heart. Numerous transitional forms can be observed between these two extremes.

In acute heart weakness due to other causes, the development of disturbances of circulation is gradual and usually does not attain a high or threatening degree until after several days. Such an evolution, however, sometimes also takes place following overexertion, if the overexertion is not excessive but occurs repeatedly. On the other hand, acute heart weakness may exceptionally develop even without previous overexertion, frequently following malignant foot-and-mouth disease in particular.

In light cases the disturbances in the circulation disappear in a few hours or days without leaving a trace and without having caused the formation of edema in the meantime, while otherwise in severe cases that are not rapidly fatal it leads to the chronic form of heart weakness, thereby as a rule losing more or less of its severity.

As Bergkrankheit is designated a condition similar to overexhaustion which often develops in man and also in animals, such as horses, mules, asses, camels, dogs, cats, and chickens if they are taken to high altitudes (more than 3,000 meters above sea level). The pathological condition was particularly observed by Fischer in pack and draft horses on the high plateaus in Bolivia and on the heights of the Cordillera; furthermore, by Gass in three army horses on duty in the mountains (Alps) and described by the latter author as "congestive conditions in horses at work in the mountains." Animals coming from the lowlands are almost exclusively affected, while animals indigenous to the mountainous regions, as a rule, do not suffer. Heavy work, cold, or intense heat, and absence of a breeze are predisposing factors. Although the degree of disturbances increases with the altitude, certain places in the regions in question are particularly dangerous, probably as the result of peculiar meteorological condition (electrical discharges in the air, radioactivity of the soil). According to the observations of Gass, horses may sicken even in relatively low regions (500 to 1100 meters above sea level) when during heavy draft or saddle work they have climbed 600 to 700 meters in a short time. The cause of the disturbance is assumed to be a decrease in the oxygen

tension of the air in the alveoli as well as the incomplete saturation of the hemoglobin with oxygen brought about by this condition. Kronecker, on the other hand, assumes as the most important basis of the disease, the impeding of the lesser circulation by the twisting of many capillaries of the lungs when the animal breathes the rarefied air. The latter view has been supported by the investigation of Lempen on the heart of calves at altitudes of 1110 to 1800 meters, 450 to 700 meters, and 30 meters. The investigations revealed a typical increase in the size of the heart and a thickening, especially of the wall of the right ventricle, of the heart of calves from the higher altitudes, due to an increased resistance in the pulmonary circulation.

The symptoms appear while the animals are at work and consist in acceleration (more than 80 per minute) and weakness of the pulse, accelerated, forced respiration, cyanosis, a medium rise in body temperature (according to Gass up to 39.6° C.), frequently hemorrhages from the nose and mouth. Furthermore an awkward, sometimes very conspicuous stringhalt-like movement of the hind legs, exhaustion and collapse are noted if the animals are not rested. Often death takes place in coma, even more frequently than in man under the same condition, because the animals are more exhausted when pulling or carrying heavy loads.

For treatment the animals are often bled; however, they also recover without treatment when rested and also soon become accustomed to the local conditions.

The symptoms of chronic heart weakness develop very gradually in the course of several days, weeks or months and at first consist of cyanosis with bluish-red discoloration of the mucosa and unpigmented skin (especially in swine), great prominence of the veins of the mucous membrane (especially of the conjunctiva), which is soon followed by pronounced filling of the jugular veins or other superficial veins, whereby the jugular veins exhibit a pronounced negative (diastolic) pulsation. This is soon followed by hydropic conditions, particularly ascites (in dogs frequently the first symptom of a hydropic condition) and later also by hydropic effusions into other body cavities. Furthermore, edema of the skin in front of the chest, under the chest, under the abdomen, on the prepuce and on the lower parts of the extremities is observed.

The insufficient supply of blood to the brain and its membranes and the venous stasis produce disturbances of the conscience and attacks of giddiness, especially during exercise and while at work. Such attacks occur very frequently, sometimes ten to twenty times a day, during which horses sometimes fall upon their carpal joints. According to Katzke this is characteristic of heart weakness during valvular disease.

The stasis of the pulmonary circulation maintains the difficult respiration and frequently leads to the development of

bronchial catarrh. A bloody or saffron-yellow nasal discharge, as a result of hemoptysis, and which exceptionally may lead to asphyxiation, is only seldom observed. Now and then epistaxis is observed and in almost all cases, pulmonary edema is the final symptom.

Symptoms of a chronic gastro-intestinal catarrh, with more or less pronounced disturbances of nutrition, frequently result from stasis of the blood in the wall of the stomach and intestines, and in the liver and pancreas. Sometimes enlargement of the liver can be detected.

According to the degree of the simultaneously present passive congestion of the kidneys, there is a varying decrease in the amount of urine. The urine is darker in color, of a high specific gravity, and now and then contains hyaline casts or a few granulated casts and red blood corpuscles.

Disturbances in the activity of the heart are never absent, particularly an accelerated, stronger and throbbing heart beat, very often an additional arhythmic systole with omissions in the always weak pulse. In severe cases, great irregularity in the activity of the heart is noted with the heart sounds occurring in volleys and with spontaneous longer pauses and other irregular differences in the size and pause of the pulse (arhythmia perpetua, delirium cordis). Besides, a dilatation of the heart can usually be detected.

By resting the animal and with proper treatment, light disturbances in the circulation disappear after a few weeks or months or show pronounced temporary improvement so that in the latter case the animal may again become useful to a certain extent. On the other hand, improvement and exascerbation of the condition may continue even without external influences.

Diagnosis. Diagnosis depends upon the peculiar symptoms of disurbance in circulation which can be recognized while the animal is at rest or only when exercised, together with changes in other organs which indicate the presence of such disturbances, whereby the condition of the organs and the behavior of the heart beat and pulse indicate the degree of the heart weakness. By a systematic examination of the heart and other organs, as well as by considering the anamnesis, one will often be able to determine the nature of the basic trouble. The elimination of disturbances in the circulation due to a vaso-motor paralysis, particularly in the course of acute febrile diseases and in poisoning, is very important. These are essentially different from acute heart weakness in that the intense engorgement of the small veins of the mucous membranes and also of the large veins is absent, that the greatly accelerated and weak pulse is not arhythmetic and respiration is not labored but simply accelerated (absence of stasis in the lesser circulation). Furthermore, a pale cyanotic discoloration of the mucous mem-

branes (particularly the mucosa of the mouth) is also recognizable as contrasted to the frequently dark bluish-red discoloration in cases of pure heart weakness. In case of a simultaneous presence of both conditions, one observes, in spite of a small or medium engorgement of the veins and less pronounced dyspnea in the absence of edema, a very great acceleration and great weakness of the pulse as well as a pronounced loss of strength in the animal. Attacks of threatening dyspnea while at work may also be produced without the presence of heart weakness by chronic pulmonary emphysema, chronic bronchial catarrh, and stenosis of the larynx (mostly paralysis of the larynx). These conditions, however, can easily be eliminated by the presence of other symptoms that usualy accompany them and by the fact that during exercise the activity of the heart is changed similarly as in healthy animals subjected to similar conditions. In cases of pronounced enemia and in very emaciated animals that have been weakened by other conditions or scanty nutrition, a pronounced acceleration of the pulse and respiration and a rapid tiring-out during exercise are also observed, but arhythmia or, if present, an increase in the same, as well as engorgement of the veins and pronounced dyspnea are absent.

Prognosis. This depends upon the cause of heart weakness, since light cases of insufficiency following over-exertion or occurring in the course of diseases from which the animals recover in a short time, usually promise complete recovery of the animal. The degree of disturbance in the circulation is of great significance, as an unfavorable termination may be expected if grave or rapidly increasing symptoms of insufficiency are present. The sooner proper treatment leads to improvement and the longer the improvement continues, the more favorable is the prognosis; one must, however, consider the kind of work the animal is to perform.

Treatment. The animal should be well fed with non-irritating feed (especially carnivora) which should be given frequently and in small quantities. One should guard against constipation and digestive disturbances and in case these do occur, they should be relieved by non-irritating purgatives (neutral salts, castor oil, calomel, rheum, aloes, etc.). Under certain circumstances, complete rest in a well ventilated stall (blanketed if necessary), or some other shelter is indicated and may suffice to remove the disturbances in circulation in light cases of heart weakness due to over-exertion, while otherwise, and also in heart weakness due to other causes, digitalis or strophanthus should be used. With these drugs one achieves a decrease in the rate of the heart beat and therewith a better diastolic pulse, besides an increase in the amount of blood propelled with each contraction of the heart. Furthermore, there

is obtained a stronger contraction of the heart and also of the abdominal blood vessels supplied by the splanchnic nerves, together with a dilatation of the peripheral blood vessels and those of the kidneys. Thus we get a better emptying-out of the venous system, a decrease in the venous blood pressure, a better distribution of the blood throughout the vascular system, a better supply of blood to the heart muscle, an increased recuperation during diastole, an increased tonus, and hand in hand with it, an increase in the initial tension of the heart muscle which in turn determines the amount of blood expelled with each heart beat, and a better elimination of waste products from the blood by the kidneys. The degree and origin of the heart weakness, the species of the animal, and the condition of the digestive organs should be carefully considered in choosing the drug and in determining the mode of administration and the dose. In case of stomachal administration of digitalis and strophanthus preparations, *their action will not take effect* until 12 to 16 or 16 to 24 hours later and only reach the desired degree of action during the following days. In case of great heart weakness with threatening symptoms, such as complete irregularity of heart action, thread-like pulse, great dyspnea, edema of the lungs, and general weakness, it is advisable to inject the proper cardiac stimulants intravenously giving one or at most, two injections at an interval of 24 hours. In this case, the action will begin within a few minutes and will have fully developed within an hour. Under such circumstances caution is necessary and one should give only one-half or one-fourth of the full dose recommended below as the already injured heart muscle is very sensitive to cardiacs and the injection of a full dose may bring about the death of the animal. In the case reported by Marek, the horse died immediately following intravenons injection of 2 mg. strophanthin. Physicians have reported several such cases. Intravenous injection should not be resorted to in case the animal has just previously been treated with digitalis or strophanthus. After the therapeutic effects of the treatment begin to become manifest, treatment may be continued with intravenous injections at intervals of two to four days, but it is better to follow up the advantage obtained by continuing the stomachal or subcutaneous administration. In cases of less threatening heart weakness as well as in all light cases, one can administer the proper cardiac subcutaneously or administer any cardiac stomachally. In the choice of the preparation and the mode of application one should consider the species of the animal to be treated, as in the horse subcutaneous injections of digitalis and strophanthns preparations cause painful swellings at the point of injection that persist for a long time and which will sometimes suppurate or become necrotic even when the point of injection is massaged immediately after the injection, as is recommended by Dorn.

Carnivora and cattle, on the other hand, tolerate subcutaneous injections very well. This fact is of great significance in ruminants, since, according to Salvisberg, the active glucosides are destroyed by the ferments in the fore-stomachs and accordingly the internal administration of digitalis is very unreliable even when the drug is given in solution or in suspension and administered so slowly that it will pass along the esophageal groove directly into the fourth stomach. Furthermore, digitalis preparations are said to cause abortion in pregnant cows. If gastric disturbances are present, the use of galenic preparations should be avoided so as not to aggravate the disturbance by the saponins and tannic acid compounds present in such preparations. Intramuscular injections may be used in the dog, but these can also be displaced by subcutaneous injection in order to avoid necrosis of the muscles.

Regarding the continuation of the treatment with cardiac stimulants, the suggestion of Fraenkel deserves full consideration. According to this, for stomachal or subcutaneous administration, the full daily dose is given but it is divided into several smaller, single doses for the first three days. Thereafter the treatment is continued with one-half of this dose until the pulse becomes stronger and decreases in frequency and an increase in the amount of urine becomes manifest. Depending upon the condition of the circulation and the behavior of the animal toward the drug, treatment may be continued with one-third or one-fourth the original dose and for any length of time. On the other hand one may give the animal a rest for one to two weeks after the animal has been treated with cardiacsforperiods of 7 to 14 days, at first with full doses and then with half doses. If necessary, one may administer caffein or other similar drugs during such periods of rest. These precautions are necessary on account of the cumulative action of all digitalis and strophanthus preparations which, however, varies to some extent and is less marked for strophanthus. In case one or the other drugs does not prove sufficiently active for the case in hand, it may become necessary to switch to the other drug.

It is very important to use only properly standardized drugs with a known amount of the active principle so that the dose and the desired effect can be properly gauged. Since the action of folia digitalis varies between 100 to 275% according to the origin, time of harvest, and manner of preservation, and since during storage the amount of the active principle may be reduced by one-half through the action of ferments, the crude drug has recently been marketed only after it was dried in a special manner and properly titrated on frogs. Furthermore, by removing the ferments and other vegetable compounds from folia digitalis and semen strophanthici, and by titrating them on the frog, stable drugs of constant action have been prepared. The stable and active glucosides have also been pre-

pared in the pure state. These drugs, however, do not represent a substitute for good folia digitalis since they do not contain the separate active glucoside in the same proportion as the crude drug (according to Straub in fully active folia digitalis, gitalin [easily soluble in cold water, decomposed by hot water and alcohol], digitalein [insoluble in water and alcohol], digitoxin [insoluble in cold and hot water, soluble in alcohol, chemically stable]). The following preparations of digitalis may be used. Folia digitalis (also obtainable as folia digitalis titrata) which is administered in powder form (horses 2 to 4 gm., cattle 4 to 6 gm. per day) immediately after feeding; to dogs as an infusion (1 to 2 gm. to 200 gm. of water, of which give 2 to 3 tablespoonsful daily) and if necessary combined with potassium nitrate or acetate, preferably with diuretin, agurin, theocin or theophyllin. Tinctura digitalis titrata (horses and cattle 10 to 25, sheep and goats 5 to 10, hogs 2 to 5, dogs 0.5 to 1.5 and cats 0.1 to 0.2 gm.) is preferable to the common tincture of digitalis with uncertain action. Digitalon (physiologically titrated dialysate, also intravenously) is said to act equally good. Extractum digitalis foliorum fluidum is also marketed in a physiologically titrated form (to be administered in 5 times the dose of the leaves at the most). According to Kunz-Krause and Roeder, folia digitalis dialysatum (for large animals 5 to 15 gm. per os) can also be used, as can digitalysatum Buerger (for subcutaneous and intravenous injection in ampules; dogs 1 to 1.5 cc.) as well as digipuratum Knoll (obtainable in powder form, in tablets with 0.01 gm. digipuratum, and in ampules with 1 and 10 cc. sterilized solution of 0.01 gm. and 0.1 gm. digipuratum equal to 0.01 and 0.1 gm. folia digitalis). It is also given internally in tablet form (dogs one-half to one tablet), subcutaneously (dogs 0.25 to 0.5 cc., horses and cattle 10 cc.), or intravenously (one and one-half to two times the cubcutaneous dose). Digifolin (in tablets and as a sterile solution in ampules; *same dose as digipuratum*) and liquitalis Gehe (same dose as digipuratum) are similar preparations, as are probably also digitan (in ampules of 7.5 cc. each for large animals for subcutaneous or intravenous injection), digitotal (in powder, in tablets or in ampules), digiclarin (in tablets equal to 0.5 gm. fol. digitalis or in solution of 1 cc. and 10 cc. equal to 0.1 and 1 gm. fol. digitalis), verodigan (gitilan in tablets equal to 0.1 gm. fol. digitalis or in solution) and digityl (a fluid extract). Digistrophan consists of digitalis and strophanthus (2:1) and is marketed as digistrophanum purum (in tablets with 0.1 gm. titrated fol. digitalis and 0.5 gm. strophanthus). It is also marketed in tablets as digistrophanum diureticum I (with 0.2 gm. sodium acetate in tablets) and digistrophanum diureticum II (like the former plus 0.15 gm. caffein). Langhoff recommends for smaller and warm blooded horses 10 to 20 tablets of digistrophanum diureticum, for medium sized horses 25, and

for heavy or cold blood horses 25 to 30, repeated three times if necessary. A preparation consisting of more or less pure glucosides is digalen Cloetta (in ampules of 15 cc., tablets of 0.5 and 7.5 cc. for internal administration and ampules of 1 cc. for subcutaneous and intravenous injection) of which horses receive subcutaneously or intravenously 5 to 15 cc., cattle 20 cc., dogs 2 to 5 cc. Adigan Richter is a similar preparation which can be obtained in tablets of 0.1 gm. and as a 30% alcoholic solution in 15 cc. ampules for internal administration; furthermore, in ampules of 1 cc. in sterile solution for subcutaneous and intravenous injection (1 tablet or 1 cc. of the solution is equal to 0.1 gm. fol. digitalis; dose same as digipuratum). The use of purified digotoxin Merck for therapeutic purposes can hardly be recommended, because its toxic action lies close to that of therapeutic action and because it immediately causes a general contraction of the blood vessels with pronounced increase in aortic pressure which may become dangerous in case of severe injury to the heart muscle. As reliable strophanthus preparations (difference in the action varies from 100 to 330% for strophanthus seeds and from 400 to 600% for the tincture) can be named the tinctura strophanthi titrata (1 to 3 drops internally for dogs) and strophanthon (solution of the active substances of strophanthus; 3 to 4 fold the dose of folia digitalis at the most), but especially strophanthin which can be obtained as strophanthinum parissimum Merck (pseudostrophanthinum Feist) and as strophanthinum Boehringer, which is easily soluble in water and alcohol and of which horses may be given 0.0025 milligram per kg. body weight (in all 0.001 to 0.002 gm.) and dogs 0.002 to 0.003 milligram per kg. body weight (in all 0.0002 to 0.001 gm.) subcutaneously or intravenously. Strophanthin Boehringer can also be obtained in a sterile solution 1:1000. A little weaker and less certain is the action of convallaramium (glucoside Convallaria majalis) because it is readily excreted and decomposed. The same is true of Convallaria majalis (besides convallaramin it also contains convallarin which is locally very irritating) as an infusion (5:150, for dogs a tablespoonful to a teaspoonful every 2 hours); furthermore, extractum convallaria rhizon. et rad. fluidum (titratum) in doses of 0.03 to 0.1 cc. for dogs.

In case of heart weakness with simultaneous relaxation of the blood vessels as is not infrequently observed in the course of severe infectious diseases, also in many cases of poisoning, and sometimes in cases of over-exertion, cardiatonic drugs alone will not suffice, but drugs constricting the blood vessels must also be used. The drug especially indicated is caffein (caffein natrio-benzoicum or natrio-salicylicum for large animals 6 to 8 gm. internally, 3 to 5 gm. subcutaneously every 6 to 8 hours); furthermore, camphor (oleum camphoratum, subcutaneously 20 to 50 gm. for large, 4 to 10 gm. for small animals; 1 to 2 gm.

for dogs, but also in watery solution intravenously); likewise ether (subcutaneously 10 gm., 5 gm. and 0.25 to 1 gm. at one dose); barium chloride (0.05 to 0.1 gm. intravenously for horses); eserine (0.02 gm. subcutaneously every 2 to 3 days for the horse); sometimes also adrenalin (tonogen, suprarenin, paranephrin). In acute paralysis of the heart due to a temporary breakdown of the nervous apparatus of the heart muscle in anemia, poisoning, etc., physicians make intracardial injections of adrenalin strophanthin or of adrenalin (Guthmann, Frenzel).

A medium amount of blood drawn from the jugular vein to relieve the load of the right side of the heart may be advantageous in cases of pure heart weakness, especially if severe stasis in the pulmonary circulation is present. Somewhat disquieting palpitation of the heart is combated by cold compresses in the region of the heart, or by subcutaneous injections of morphine. According to Lienaux, a troublesome cough with a simultaneous bronchial catarrh can be relieved with apomorphine (0.005 to 0.05 gm. for large, 0.003 to 0.005 gm. for small dogs). In case of hydropic conditions calomel may be tried (for dogs 3 to 4 times daily 0.05 to 0.1 gm. alone or with the same amount of oleoresin jalapæ), but diuretics (potassium acetate and nitrate, scilla) give more promise of success. Furthermore one may use agurin (for dogs 1 to 3 gm. daily in water), theocin (same amount), theophyllin (same) and diuretin (same), especially if the consumption of water and sodium chloride is restricted at the same time. Periodic tapping for the removal of transudates from the body cavities can sometimes not be avoided. The operation should, however, not be performed if the transudate soon recurs.

Acute Disturbances in the Circulation Due to a General Relaxation of the Blood Vessels. (Paralysis of the Vaso-motor Nerves.) A relaxation of all small arteries does not take place only under the influence of certain chemical substances (amyl nitrite, alcohol, chloral hydrate, chloroform) when they gain access to the animal body in large quantities, but also as a result of toxins in various infections as was shown experimentally by Romberg, Bruhns, Baessler and Rolly. A relaxation of the blood vessels occurring in a natural way and in varying degrees is a very common occurrence in the course of various specific, as well as ordinary acute diseases, where it forms the sole or principle basis of the commonly present disturbances of circulation, aside from an accelerated and correspondingly weak pulse due directly to the fever. Under such conditions the relaxation of the blood vessels may combine with a degeneration or inflammation of the heart muscle. The paralysis of the vasomotor nerves becomes especially prominent and assumes a severe degree in case of sudden inundation of

the blood with infectious material, toxins, or certain chemical poisons as in rupture of the stomach or intestine, rapidly developing gastro-enteritis, peritonitis, and in certain poisoning (arsenic, antimony, heavy metals, cantharides).

Under the influence of the above mentioned toxic substances, a general relaxation takes place, first appearing in the region of the splanchnic nerves. Soon, however, and in severe cases from the very onset, a decrease in the tonus of the peripheral blood vessels develops, as a result of which the very elastic blood vessels of the abdominal organs, which can not be subjected to any great pressure by the tissue and which, especially in herbivorous animals, are very cavernous, take up the greater part of the total volume of the blood of the animal and in this manner bring on anemia of the other organs. The animal bleeds to death, so to speak, into its abdominal blood vessels. This causes a sinking of the blood pressure and a decrease in the velocity of the blood, with a decrease in the fill of the peripheral blood vessels, and following this, a corresponding acceleration of the heart.

Symptoms. Aside from the symptoms of the basic disease, one always observes a usually pronounced acceleration, weakness and softness of the pulse without arhythmia. Furthermore, there is cyanosis with a bluish, pale (livid) discoloration of the visible mucous membranes (mostly only recognizable on the mucous membrane of the mouth, because the conjunctiva or the nasal mucosa are frequently reddened due to the fever). A stronger engorgement of the veins is absent. Depression and weakness with swaying and staggering gait, corresponding to the degree of the circulatory disturbances and acceleration of the respiration, are also a part of the clinical picture of paralysis of the vasomotor nerves, which, in severe cases, terminates fatally in a short time.

Regarding a differential diagnosis, true heart weakness must be eliminated.

Treatment. An incomplete paralysis of the blood vessels can be relieved by intravenous or subcutaneous injection of drugs constricting the blood vessels, repeated every five or six hours, for which purpose, besides caffein, the suprarenal preparations deserve consideration for severe cases, provided a simultaneous heart weakness can be excluded. Their action can be supported with peripheral irritants (rubbing with drugs, slightly irritating the skin, cold rubs), and under suitable conditions inflation of the udder with air. Furthermore, intravenous infusion of physiological salt solution or one of the artificial sera is indicated in quantities similar to those used in the treatment of acute anemia. If necessary, subcutaneous injection of such solutions may have a favorable action.

5. Dilatation of the Heart. Dilatatic cordis.

The term incompensatory, myogenic or stasis dilatation of the heart is used to designate an increase in the capacity of the heart cavities due to insufficient systolic emptying of the same with a relative or absolute decrease in the power of the cardiac muscle. Adaptatory dilatation, on the other hand, is due to an increased diastolic filling of the cavities lying centrifugally, with insufficiency of the valves (compensatory dilatation) and to an enormous inflow of venous blood during violent muscular contractions. In the latter case, a systolic dilatation develops simultaneously in spite of a decrease in the amount of blood propelled at each contraction (accommodative, tonogenic dilatation, acute dilatation due to exertion). The latter form disappears when the muscular contractions cease and this form, as well as compensatory dilatation, will not receive further consideration at this time.

Etiology. In general, the etiology coincides with that of heart weakness, as in relative as well as in absolute heart weakness, the considerable amount of blood remaining in the cavities after each systole and the excessive diastolic expansion of the weakened heart muscle, easily lead to a permanent increase in the lumen of the heart cavities. Accordingly, a dilatation usually follows heart weakness, especially if the latter is of a long duration or rather pronounced. Schmidt repeatedly observed in horses a dilatation of the heart with relative insufficiency of the valves as a result of insufficient nutrition while the animals were doing hard work during the war. In young dogs, we have repeatedly observed a strumous heart with pronounced dilatation (also reported by Wilms in artificial struma). According to the assumption of physicians, these cases have besides a probable compression of the trachea, an increased secretion of the hyperplastic thyroid gland which has a permanent stimulating effect upon the heart action, as in Basedow's disease ("cardiovascular equivalent of complete Basedow syndrome"). According to the manner of development of the heart weakness, one can distinguish between acute and chronic dilatation of the heart.

Anatomical Changes. The affected cavities of the heart are larger than normal. The walls are flabby, so that the opening is not patent when an incision is made on autopsy. If the size of the heart was normal before the development of the dilatation, the walls are thinner in proportion to the degree of dilatation. If the disease has lasted for some time, there occurs an atrophy of the muscles, which may be so extreme that the visceral layer of the pericardium comes into opposition to the endocardium (Dilatatio passiva). If, on the other hand, the dilatation has developed in a heart that had previously become hypertrophied, the wall of the dilated cavity either remains of normal thickness, or appears to be even thicker (Dilatatio activa). In this case the presence of

dilatation may be surmised, aside from the diameter of the respective cavities, by the fact that the trabeculae on the inner surfaces of the ventricular wall are narrow and separated one from the other by wide spaces. At the same time the chordae tendineae are usually thin and elongated. The muscles almost always show signs of parenchymatous or fatty degeneration and also of atrophy. In other organs changes due to stasis, together with dropsical effusions, exist.

Symptoms. In severe cases, enlargement of the cardiac dullness may be shown, which is sometimes very considerable, *while in animals in which normally an absolute heart dullness* is not found, above all the occurrence of such an absolute heart dullness is noticeable. In carnivora the dilatation of the left ventricle causes an enlargement of the cardiac dullness

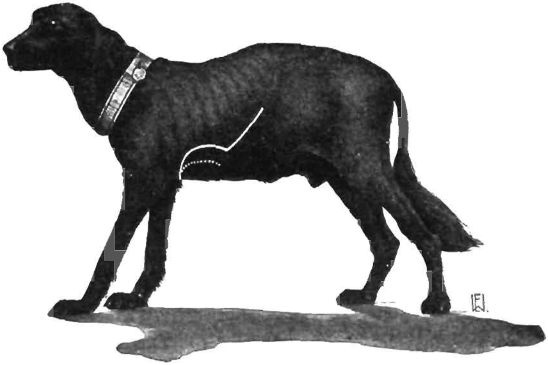

Fig. 152. **Cardiac Dilatation.** The dotted line gives the normal heart dullness, the solid line indicates the enlarged heart dullness (4th to 6th intercostal space), the dullness passes backward into the anterior border of the liver dullness, which is *also enlarged.* (Same dog as in Fig. 153.)

upward beyond the mammary line (Fig. 152); in other animals, both upward, and even more *backward (Fig. 154).* On the other hand, the dilatation of the right ventricle is characterized by the occurrence of an absolute dullness on the right side, or by an enlargement of the dullness which is normally very slight in small-chested animals. (Figs. 153 and 155.) Considerable dilatation of the right *ventricle, however, causes an increase* of the dullness even on the left side. It is possible, as shown by a case of the author's in a dog six weeks old, for the cardiac dullness in dilatation of the right half of the heart to extend forward as far as the second intercostal space. It is not certain

whether this is to be observed only in very young animals. The force of the heart beat is usually increased because a greater portion of the heart is in apposition to the chest wall. Sometimes the trunk receives a thrill synchronously with the heart beat. The beat appears weaker only when the heart weakness has become considerable. The systolic heart sound is increased in force like the heart beat, while the second sound is usually small or not at all perceptible. The action of the heart is not infrequently arhythmical, and the first, less often also the second heart sounds are dicrotic. Endocardial sounds are caused by the relative insufficiency of the valves which develops after a while in some cases.

In contrast to the increased heart beat, the pulse is always feeble. It may also be arhythmical and intermittent, corresponding to the heart beat.

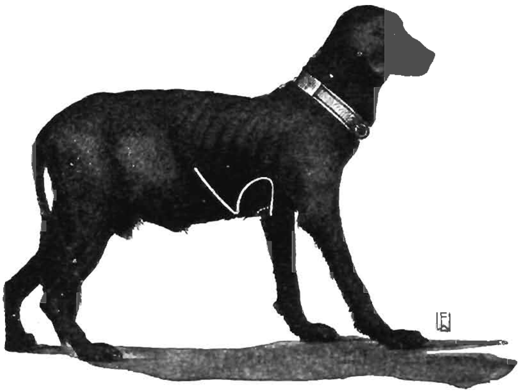

Fig. 153. **Cardiac Dilatation.** Enormously increased cardiac dullness on the right side. The dotted line gives the borders of the dullness, which occurs normally in large dogs. (The same dog as in Fig. 152.)

The dilatation of the weakened heart is a compensatory phenomenon, since in an increased diastolic filling the initial tension of the heart muscle is increased, thus enabling it to increase the amount of blood propelled at each contraction as contrasted to the initial condition of the heart weakness without dilatation. Nevertheless in such a heart the residual blood increases as contrasted to the normal amount in an unchanged resistance and increasing fill, as well as in unchanged fill and increasing arterial resistance.

In a rooster with pronounced dilatation of the right ventricle which became about the size of a hen's egg, Reinhardt observed dyspnea from compression of the trachea, enlarged abdomen and emaciation. The cause of the dilatation proved to be due to the emptying of the hepatic vein directly into the right auricle and a non-closure of the ductus Botalli.

Dilatation of the left ventricle, as the result of muscular degeneration which is often accompanied by hydrothorax, is frequently observed as the main lesion in so-called gauwziekte. This disease is due to an intoxication from eating the plant *Vangueria pygmaea* found in South Africa and usually terminates fatally in four to six weeks or even later. Only plants growing on wet soil containing iron are dangerous.

Course. If the animals are cared for, the symptoms may, if they are not yet severe, diminish considerably, and in such cases the cardiac dullness will become much smaller. On the

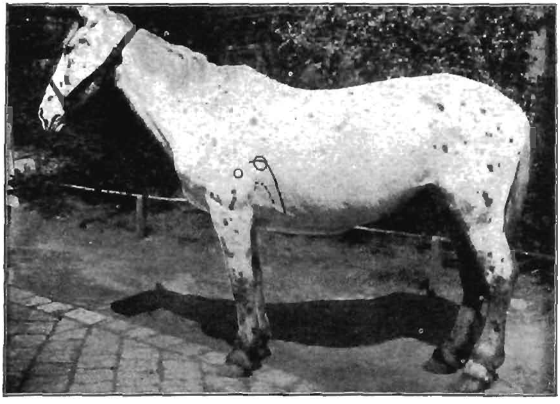

Fig. 154. **Cardiac Dilatation.** The dotted line gives the borders of the normal heart dullness, which appears in the illustration to lie too high, on account of the swelling of the lower chest. The solid line indicates the upper and posterior borders of the greatly enlarged heart dullness. Owing to the dilatation, the punctum maximum of the systolic sounds arising in the left venous ostium (large circle) and of the second pulmonary sound (small circle) is displayed upward. (Compare Fig. 155.)

other hand, phenomena of stasis of a decided character are frequently absent in a strumous heart, in spite of pronounced dilatation, while psychic excitement, as well as relatively slight movements of the body, may cause threatening asphyxiation or

immediate death of the animal. Acute dilatation may even terminate in complete recovery.

Diagnosis. The characteristic symptoms of cardiac dilatation are: enlargement of the heart dullness, increase in the force of the first heart sound, arhythmia of the heart action, feeble pulse, symptoms of stasis. The feebleness of the pulse differentiates the disease from primary hypertrophy of the heart. The absence of friction sounds distinguishes it from pericarditis or from hydropericardium.

Treatment. Permanent recovery may be hoped for only if a previously healthy heart has become dilated owing to very severe muscular exertion. In such cases the circulatory disturbances retrogress considerably or entirely under absolute rest and through the influence of repeated administration of cardio-tonic remedies, first of all digitalis. This improvement may occur in a short time. (For details see the chapter on

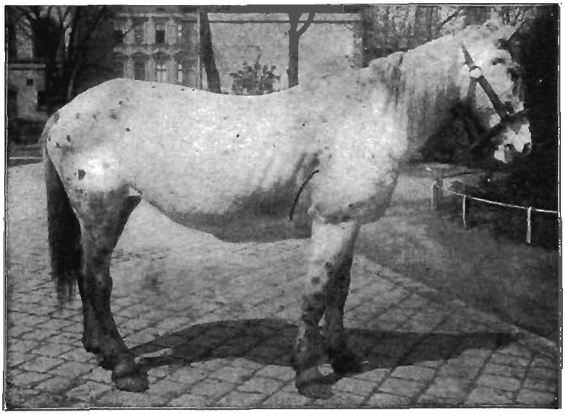

Fig. 155. *Cardiac Dilatation.* Heart dullness of the horse pictured in Fig. **154**, on the right side, in the 4th to 5th intercostal space: in front the upper border reaches the scapular line.

valvular diseases.) Even dilatation which commences in the course of acute infectious diseases may sometimes be reduced by similar treatment; but if the heart had been diseased previously or if there are permanent impediments to the circulation of the blood, if, moreover, signs of degenerative processes

exist, it may be possible to obtain an improvement that is at times quite considerable, but this will soon yield to an aggravation of the condition. Such patients can be used for work only to a very limited degree. It is always necessary to insure nutritious feeding and to save the animals from work.

In so-called strumous heart one may, under certain circumstances, try galvanization of the cervical sympathetic nerves and also sedative drugs; in case of symptoms of stasis use also cardiac stimulants.

6. Hypertrophy of the Heart. Hypertrophia Cordis.

Hypertrophy of the heart consists in an increase in the heart muscle which arises from thickening and possibly from *multiplication* of the muscular fibers under the influence of an increase in function persisting for at least four weeks.

Etiology. During severe work the normal, or usually increased blood is forced against the normal or increased resistance at greatly shortened intervals, and if this is repeated time and again it is followed by hypertrophy of the heart muscle. In horses used for rapid motion, in hunting dogs, and exceptionally in pack-horses and draught-oxen, the relatively great size of the heart stands in relation to the increased muscular exertion. A comparatively large heart may be said to be a constant finding in some species of these animals, and it is not improbable that heredity may be of influence upon the dimensions of the heart. This physiological hypertrophy is always in proportion to the development of the muscles of the body and does not require any treatment.

On the other hand a pathological hypertrophy of the heart does not stand in direct relation to the development of the skeletal muscles or is limited to some portions of the heart. In persistent disturbances of the circulation the part of the heart which is connected with the place of the disturbance is forced to continuously increased exertion and this is eventually followed by hypertrophy of this portion of the heart; in diseases of the left venous ostium, however, the right ventricle also hypertrophies owing to increased work done. Pathological processes in the heart muscle itself, such as chronic myocarditis, tumors, echinococcic cysts, etc., also lead to an increase in the muscular tissue in those parts of the heart which are not immediately affected. According to the experiments of Stroebel, even a pronounced stenosis of the trachea may cause hypertrophy of the heart. A similar result is produced by adhesions of the layers of the pericardium because it necessitates an increased amount of work on the part of the heart. Acute cardiac dilatation may also form a basis for the development of hyper-

trophy if the heart muscle is comparatively unimpaired. (In chronic myocarditis the hypertrophy may at times be only apparent being simulated by the fact that the wall of some portion of the heart has become thicker owing to cellular infiltration or to simultaneous new-formation of connective tissue [E. Albrecht]). Finally hypertrophy of the heart often develops in older dogs in consequence of a chronic, especially interstitial, inflammation of the kidneys.

The secondary hypertrophy of the heart which occurs in connection with chronic diseases of the heart, lungs, arteries or kidneys is a phenomenon of these diseases and will therefore be considered in connection with them. Here only the so called idiopathic hypertrophy of the heart is to be discussed which arises from an acute cardiac dilatation or is possibly produced by irritating substances that are not known exactly.

Anatomical Changes. The determination of cardiac hypertrophy requires an exact knowledge of the physiological dimensions, the relations between heart and body weight and a consideration of the size of the respective heart cavities.

The relation of the weight of the heart to the weight of the living body in the horse is, according to Franck & Martin, 1% on an average; according to Frey, 1.28%; according to Schubert, 0.84%; in cattle, according to Schmalz, 0.33-0.42%; according to Schneider, in the steer 0.442%, in the ox 0.414%, in the cow 0.492%; according to Schubert, in the steer 0.436%, in the ox 0.42%, in the cow 0.52%, and in calves 0.708%; in the dog, according to Schubert, 1% of the body weight.

During moderate contractions the hypertrophy of the muscle may be noticeable even on external inspection, especially if it is only partial. The form of the heart is changed thus that with an increased thickness of the left ventricular wall the heart as a whole is slender, elongated, like a pointed cone, while in hypertrophy of the right ventricular wall the heart as a whole becomes broader and more flat, the right heart being involved in the formation of the apex; indeed, if the hypertrophy is very great, the right ventricle reaches beyond the apex of the left ventricle. The thickened muscle is usually firm and like rubber. The weight of the heart is also increased and may in well marked cases amount to twice the normal weight.

The customary classification of simple hypertrophy (walls thickened, cavity of normal width), concentric hypertrophy (walls thickened, cavity smaller) and eccentric hypertrophy (walls thickened, cavity dilated) is well adapted for a concise description of the findings, but it has no particular significance, except that the last form indicates a considerable insufficiency of the previously hypertrophied heart muscle.

Symptoms. Physiological hypertrophy of the heart does not give rise to any morbid symptoms. The symptoms of hypertrophy which develop in connection with organic heart

diseases or arterial affections will be considered in connection with these diseases.

In the remaining cases the hypertrophy is characterized by an enlarged cardiac dullness, increased force of the heart beat, accentuated diastolic heart sound and by a strong and tense pulse.

If the energy of the heart contractions is diminished later on, the symptoms of cardiac insufficiency appear which correspond with those of dilatation (p. 764). At first they usually occur only on motion, but may later be observed also at rest. There are then noticeable languor, feeble pulse, increased fullness of the veins, gradually increasing emaciation, dropsical signs, sometimes vertigo and attacks similar to palpitation.

Treatment. The treatment will have to be limited to preventing, if possible, the exhaustion of the heart muscle; this is done by careful use and proper feeding of the patients. The animals may, even for considerable periods, be used for uniform and quiet work, but exertion usually decidedly aggravates their condition, the circulatory disturbances are increased and sudden death may follow. In cardiac weakness cardiotonic remedies are indicated (see valvular heart defects).

7. Inflammation of the Heart Muscle. Myocarditis.

(a) Acute Myocarditis. Myocarditis Acuta.

There are two principal types of acute inflammatory processes in the heart muscle, namely, the parenchymatous myocarditis, which is characterized by a pathological effusion that may be very slight and by a more emphatic degeneration of the muscular fibers, and secondly, purulent myocarditis, in which purulent softened foci are developed in the heart muscle.

Etiology. Aside from the inflammation, which, according to some authors (Friedberger & Fröbner, Zschokke) develops primarily in exceptional cases in horses and hunting dogs owing to excessive exercise, acute parenchymatous myocarditis is a secondary affection, which occurs especially in acute infectious diseases and very rarely in certain intoxications. Such cases were probably instances of acute dilatation with pure degeneration, like the not infrequently occurring myocarditis in calves, following long transportation by rail as reported by Zschokke. The toxins which, in the course of acute infectious diseases, circulate in the blood, produce sometimes only a parenchymatous degeneration or at the same time a fatty degeneration of the muscular fibers, sometimes also a serous or cellular infiltration of the interstitial tissue, and frequently hemorrhages. This occurs especially in the septicemic diseases, for instance, in hemorrhagic septicemia, in swine erysipelas,

anthrax, and acute glanders, while foot and mouth disease and especially variola, seem to be complicated by myocarditis in their malignant forms only. Acute myocarditis occurs in the course of malignant foot and mouth disease, mostly when the lesions on the mucous membrane are in the stage of healing, especially in herds of swine. It sometimes develops without the formation of vesicles in all or most of the animals. In such cases, where numerous deaths may occur, the only proof worthy of notice in the detection of the cause is furnished by the peculiar focal lesions of the heart (tiger heart) and should remind one to try transmission experiments, if necessary. Lesage observed enzootic myocarditis accompanied by foot and mouth disease in a flock of lambs. The disease was presumably introduced into the flock by a breeding ram.

An inflammation which was observed by Dieckerhoff in connection with the so-called skalma and which was called by him "myocarditis septica," undoubtedly develops upon the basis of an influenzal infection. Since in infectious diseases the heart muscle behaves like a parenchymatous organ, the frequency of myocarditis in infectious diseases may best be indicated by the comparatively frequent occurrence of renal albuminuria, which points to a parenchymatous disease of the kidney.

Of poisons, especially the heavy metals (mercury, copper, antimony), also phosphorus and arsenic are capable of causing a degeneration of the heart muscle.

Myocarditis, usually purulent in character, develops if infected emboli have been carried into the blood vessels of the heart (myocarditis embolica), which occurs most frequently in cattle. The emboli may be derived from any part of the body where suppuration or gangrenous disintegration of tissues is present, but most frequently a purulent or gangrenous inflammation of the lungs or of the intestinal tract, a disintegrating thrombus of the umbilical vessels, purulent inflammation of the hoof, puerperal endometritis, strangles, glanders or decubital gangrene are responsible for the affection. In cattle the necrosis bacillus sometimes gives rise to the formation of dry necrotic foci in the heart muscle. Furthermore, Singer found a purulent actinomycoma in the heart muscle of an ox.

Injuries of the heart muscle produce a purulent or ichorous myocarditis most frequently in cattle and in the same manner as the inflammation of the pericardium which has usually developed earlier from the same cause (see page 742). It is much less frequent that foreign bodies which have penetrated the wall of the thorax and have entered the heart produce myocarditis, because such injuries usually cause death even before an inflammatory process could possibly be developed.

Anatomical Changes. The affected heart muscle appears pale reddish-brown or gray red, dull, flabby, tears easily, and

discloses on the cut surface dark red spots, or on the contrary light gray or even butter-yellow points and streaks (myocarditis acuta multiplex). Especially striking are such lesions in the heart muscles in animals which died suddenly from the malignant form of foot-and-mouth disease, where Joest observed them in the wall of the left ventricle and its septum, but in no instance in the wall of the auricles, but frequently saw very closely placed subepicardial hemorrhages. Adult cattle disclose in these lesions round cells and granulation tissue principally, with a considerable degeneration of muscle fibres; in calves, however, severe parenchymatous changes predominate (cloudy swelling, granular and fatty degeneration), according to Nieberle, partly a purulent focal degeneration with only slight reaction of the very vascular connective tissue and partly a degeneration with pronounced inflammation. In other cases of acute myocarditis, however, the microscopic examination may reveal swellings, granular cloudiness, sometimes also fatty degeneration, and in severe affections granular degeneration of the muscle fibres (Zschokke). In myocarditis in calves resulting from railroad transportation, Zschokke found a patchy or diffuse grayish-white discoloration of the surface of the heart. Upon section, he observed circumscribed spots or similar extensive subepicardial and subendocardial discoloration of the heart muscle, with granular and parenchymatous degeneration of the muscle fibers.

Metastasis or immediate continuation of a purulent inflammation from the vicinity leads to the formation of smaller pus foci which form abscesses by gradual increase in size or by confluence (myocarditis purulenta). The contents of an abscess may eventually be inspissated into a cheesy mass and even become calcified. It may also break into the pericardial cavity or into a heart cavity. Occasionally ichorous pus is found in the abscesses. (In the heart muscle of calves which were apparently healthy Kitt found repeatedly a diffuse necrosis which was similar to the necrosis observed in hogs in the skeleton muscles.)

Symptoms. When the function of the heart muscle is diminished by somewhat extended inflammation, symptoms of heart weakness, such as pronounced acceleration of the heart, strengthening and arhythmea of the heart beat, and weakness of the pulse may be observed. This is accompanied by symptoms of stasis, such as dark red discoloration and later a cyanotic appearance of the visible mucous membranes, marked fullness of the venous branches, and prominence of the larger veins. These symptoms may again gradually retrogress; in severe cases, however, they increase in severity, and finally the pulse becomes threadlike, very irregular and intermittent, with venous pulse, attacks of vertigo, twitching, and collapse, whereupon the animals die from

paralysis of the heart. Sometimes, however, they may succumb suddenly or after a short dyspnoea without having disclosed any signs of disease on a superficial observation. Such sudden deaths occur especially in the malignant form of foot-and-mouth disease; sometimes, however, they may be observed in contagious pneumonia of horses, and also following the rupture of an abscess into the pericardial sac or into the cavity of the heart, sometimes, however, without such rupture of the abscess (Thum, Joest, Singer). In spite of this, very distinct disturbances in the action of the heart can be detected upon close examination in such cases. As Marek and recently Schmotzer and Wester have shown there is an increase in the number of heart beats in adult cattle up to 80-120-140, and in calves up to 200-240 per minute in foot and mouth disease. Furthermore, there is also a more or less distinct arhythmia, usually due to an additional systole, more seldom due to a true intermission, which may become so severe as to produce delirium cordis and not infrequently also accelerated respiration in the absence of other pronounced phenomena and without disturbance in the appetite or the behavior of the animal. Higher degrees of disturbance in the activity of the heart may lead to sudden heart-failure even after a relatively slight, additional exertion as in rising and lying down, leading out of the stall, playing in the open, or even during engorgement of the stomach, in spite of the fact that the severely affected heart was able, under the favorable influence of body rest, to maintain the blood pressure and circulation necessary for the undisturbed functioning of the separate organs.

Myocarditis in young calves following transportation by rail manifests itself, according to Zschokke, in debility, and in case the costal portion of the diaphragm is similarly affected there is also an accelerated respiration. Such calves are subjected to emergency slaughter.

Diagnosis. Acute myocarditis may be mistaken especially for acute peri- or endocarditis, since in both these diseases the heart beat is usually much stronger and bounding. In the initial stage the differentiation, on the ground of clinical symptoms alone, is hardly possible. Later on, however, these diseases may be eliminated by the absence of abnormal sounds. —The general vascular paralysis which is frequent in acute infectious diseases is characterized by the fact that the veins are not filled very strongly and by the pale blue discoloration of the mucous membranes. This affection is frequently associated with acute myocarditis.

The differentiation of suppurative myocarditis from the non-purulent form is possible at the best only in those cases in which the other symptoms are indicative of a possibility of metastasis. On the other hand, the presence of an acute, paren-

chymatous nephritis, indicates the presence of a simultaneous parenchymatous myocarditis.

Prognosis. In the prognosis of acute myocarditis, the primary affection, the number, strength, intensity, and rhythm of the pulse should be considered. Generally an unfavorable termination is expected when the increase of pulse beat exceeds twice the normal, if it shows irregularity and weakness, or if distinct signs of stasis are present. It is further unfavorable in myocarditis developing in the course of certain infections diseases (for instance, in foot-and-mouth disease). In suppurative inflammation of the heart muscles the prognosis is unfavorable.

Treatment. The cardiac weakness may be counteracted by excitants or cardio-tonics. According to the intensity of the functional disturbances, the following remedies may be considered: Alcohol (in the drinking water); for smaller animals wine or brandy, digitalis (in powder form, as infusion, maceration or dialyzation), strophanthin subcutaneously, camphor internally, or camphorated oil subcutaneously, ether subcutaneously, etc. Gmeiner always observed good effects in myocarditis following upon acute infectious diseases from caffeine (large animals 6 to 8 gms., smaller animals 0.5 to 1.0 gms.) subcutaneously every 6 to 8 hours. (For further treatment see chapter on valvular heart defects).

It goes without saying that absolute rest and good nutrition of the patient must be secured.

(b) Chronic Myocarditis. Myocarditis Chronica.

Chronic inflammatory processes in the heart muscle occur in domestic animals much more frequently than formerly was assumed. A part of the cases described as idiopathic hypertrophy or as dilatation of the heart was evidently due to chronic myocarditis.

Etiology. Chronic myocarditis not infrequently develops from the acute form. Further, abscesses developing in the heart muscle incite an increase in connective tissue, either in their immediate vicinity or in more distant portions. Occasionally chronic muscular rheumatism, chronic nephritis, chronic endoarteritis, may act as exciting causes. Tuberculous foci or abscesses with circumscribed myocarditis are sometimes seen in cattle and dogs, exceptionally also in hogs, not infrequently in chickens (Fölger). Chronic peri- and endocarditis is sometimes also followed by chronic myocarditis, either from a direct continuation of the inflammatory process or from the same cause as the peri- or endocarditis, and in fact the inflammatory process may occur simultaneously in the heart muscle. In a portion of the cases the disease undoubtedly de-

velops idiopathically for reasons that are not known. Occasionally, perhaps, certain chemical substances or mechanical influences and agencies leading to increased arterial pressure may be of importance. Fleischer & Lode have produced necrotic foci in the heart muscle in 59% of their experiment rabbits after combined injections of adrenalin and caffeine, and the necrotic foci were much smaller if adrenalin alone was injected intravenously. Finally at times the embolic obstruction of the coronary arteries may induce the formation of myelomalacious indurations in the heart muscle.

Anatomical Changes. The heart muscle is more solid, either throughout or, more frequently, in portions of various extension, and contains grayish-white spots or streaks (scars). (In extensive degeneration of the cardiac muscular fibers, the heart muscle may, however, become softer, even before the occurrence of an active tissue atrophy.) More extensive scars are bulged outward through the action of the blood pressure (aneurysma cordis chronicum fibrosum). The heart is enlarged in the case of extensive disease, both owing to a thickening of its walls and because of a dilatation of the cavities.

Symptoms. Smaller scars in the heart muscle sometimes do not cause any morbid symptoms. Since, however, larger portions of the heart are usually affected, manifestations of heart weakness nevertheless appear sooner or later. A partial atrophy of the muscular fibers may, in certain cases, be compensated by the increased functioning of the unaffected fibers, but this compensation lasts only for a time and finally the symptoms of heart weakness appear in such cases also.

In the initial stage of the disease, which occasionally lasts a long time, the cardiac insufficiency becomes evident only after severe exertion or excitement. The pulse is much increased, usually arhythmical and feeble. The heart beat is irregular and bounding; the systolic heart sound loud and high. The veins are filled strongly. There is vertigo, staggering gait, rapid fatigue, severe dyspnea, occasionally collapse. In the further course these symptoms become manifest even at rest, after a dilatation of the heart has been added in the meanwhile, owing to which endocardial sounds may be manifest. After variable periods of time serous fluid accumulates in the serous cavities and cutaneous edemata develop.

Course. The condition of the animals often varies considerably, especially under the influence of external conditions. In some cases, especially after over-exertion, death may occur suddenly even before the condition has become very serious. On the other hand, the disease may be recognized in animals which are saved from excessive exercise only on the occurrence of symptoms of stasis (chronic bronchial catarrh, dropsy,

gastro-enteric catarrh, etc.). In a case reported by Heidrich, an ox after feeding showed trembling, pronounced dyspnea and great debility. On postmortem a tuberculous focus the size of an apple was found in the wall of the left ventricle.

Diagnosis. Chronic myocarditis may be recognized only then when the development of the morbid condition out of an acute disease of the heart or of its linings can be followed exactly by continued observation of the animals. In making a diagnosis, chronic endocarditis, chronic pericarditis and chronic pulmonary affections must especially enter into consideration.

Treatment. In very ill animals which have become useless for work, no opportunity is offered for treatment, because such animals are usually disposed of otherwise. In other cases a similar therapeutic proceeding is indicated as in dilatation of the heart (see page 782).

Sequel of Foot and Mouth Disease. (Asthma cardiacum postaphthosum.) This trouble was first described by Hungarian veterinarians (Schleiffer, Viasz, Horváth, László), later also by Giovanoli in Switzerland and more recently by Dutch veterinarians and also by Hamoir in Belgium and Luxemburg. The disease is observed in adult cattle and has as its basis a chronic focal myocarditis with pulmonary emphysema and probably also a general injury to the protoplasm due to the virus of foot and mouth disease. Similarly discolored, acutely degenerated foci which occur on the myocardium are also regularly found in the skeletal musculature.

Anatomical changes found by Giovanoli and Hamoir consisted of a pronounced chronic, partly interstitial, pulmonary emphysema particularly in the apical lobes, a significant dilatation of the heart with a thinning of the wall, and besides, a tougher consistency of the yellowish discolored myocardium which is studded with whitish, sclerotic or calcified foci. In one case, Hamoir observed sclerosis of the cortex of the suprarenal glands.

The disease appears one and one-half to two months after recovery from a light or severe case of foot and mouth disease. According to the severity of the symptoms, one observes, in spite of a good appetite, a gradual ceasing of the milk flow, long stary coat of hair (see Fig. 156) which may resemble that of a bear, and greatly accelerated, labored breathing with dilated nostrils. During respiration the mouth is open and the tongue protruded, while during exercise the dyspnea may be increased to such an extent as to bring on attacks of asphyxiation. Besides symptoms of pulmonary emphysema there may be observed, even when the animal is at rest, but particularly following slight ex-

ercise, acceleration and often arhythmia of the heart beats and weakness of the pulse.

The disease always takes a very slow and unfavorable course, as only a relatively small number of the light cases recover and then very slowly. Even such animals as eventually recover remain virus carriers for months according to the observation of Dutch veterinarians, but their number can be greatly decreased by promptly treating the foot and mouth disease cases with serum.

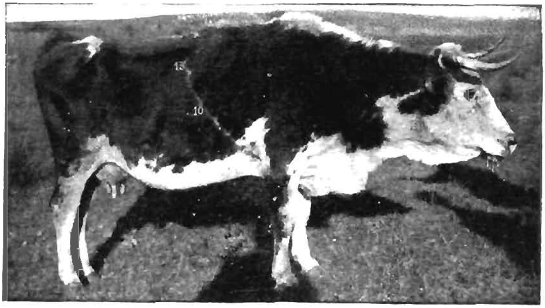

Fig. 156. Sequel of Foot and Mouth Disease. Emaciation, long shaggy coat of hair (in August), frightened look, salivation, mouth slightly opened, boundary of lungs dislocated posteriorly (see Fig 119).

Medicinal treatment (potassium iodide, cardiotonic drugs, Fowler's solution) promises no success. Only when the animals are given complete rest and good, nutritious food, light cases will make a satisfactory recovery while older, greatly emaciated animals, showing pronounced dyspnea should be slaughtered as soon as possible.

Ossification of the Heart Muscle. This is an extremely rare disease, which so far has usually been observed only in older animals, exceptionally in younger animals (by Cadiot in a five-year-old pony) and in cattle (Marty, Joest). The cause of the condition is unknown. Stoss speaks of an extensive irritation produced by great variations in the blood pressure. The ossification begins almost without exception in the wall of the right auricle. At first connective tissue proliferation occurs, then calcification and finally islands or platelets consisting of spongy bone substances. By union of these bone plates, which occurs later on, the wall of the auricle may eventually be transformed into a large connected bony plate. The process of ossification does not progress beyond the place where the veins open into the heart. The ossified auricle is usually considerably enlarged, full and tense.

In lighter cases the ossification of the auricle does not interfere *noticeably with the heart action.* If, however, the process is more extensive, signs of heart weakness become manifest during work, and later on also at rest. The cause of these signs cannot be determined during the life of the animal.

7. Fatty Heart. Cor Adiposum.

Etiology. In increased deposits of fat the heart becomes covered by a layer of fat up to several millimeters in thickness, and may, moreover, form in different places larger deposits of fat, as well as polypoid nodes on the surface of the heart. The fatty degeneration of the muscle fibers of the heart, with a degeneration of the protoplasm in poisonings and in infectious diseases, has already been discussed under acute myocarditis.

Symptoms. In an excessive degree of fatty heart, *symptoms of heart weakness present, such as feeble heart beat,* perhaps an increased cardiac dullness which, however, is difficult of demonstration in obese animals; also feeble heart sounds, possibly anorganic murmurs, feeble, small pulse; in severe cases vertigo, and finally cutaneous edema. The production of dyspnea and of vertigo is to a certain degree to be credited to the deposit of fat in the respiratory muscles. These symptoms become marked after severe exercise, or even during simple motion. Death occurs finally through paralysis of the heart, pulmonary edema or rupture of the heart.

Treatment. The treatment for obesity (Vol. III) is indicated in this condition. If signs of cardiac weakness are already present, cardio-tonic remedies must be considered.

9. Rupture of the Heart. Ruptura Cordis.

Etiology. The healthy heart muscles tear at most under the immediate influence of an injury (external injuries, falling down in cattle, from wounds caused by a foreign body entering from the reticulum). Thus Thum found in a cow a penetrating rupture of the muscular wall of the right ventricle, and a tear of about 1 cm. depth on the opposite wall of the heart, without injury to the skin or the pericardium, which had been induced by being butted in the heart region with the horns of another animal. Dull pressure exerted upon the heart can hardly produce a rupture. *Usually rupture of the heart* forms a secondary occurrence which is observed when the resistance of the heart muscle has been reduced by degeneration or atrophy. This is most frequently the case in animals with valvular troubles, where, however, the permanent increase of the blood pressure in certain portions of the heart is also of importance. Rupture of the heart can, far less frequently,

be brought into causal relation with occlusion of the coronary arteries or with abscess in the heart wall. In these conditions there usually exists already a circumscribed bulging of the diseased heart wall (aneurysma chronicum cordis) and this persists under special conditions. A bulging of the wall of the heart may occur exceptionally in acute myocarditis (aneurysma acutum cordis). As further causes for heart rupture may be mentioned tumors or parasites (especially echinococcus), in horses also aneurysm of the coronary arteries, which, however, is very rare (Walter, Gurlt, Bogmann, Cadiot, Piot-Bey).

The bursting of a heart which is already diseased occurs sometimes without any special cause, particularly under the influence of a normal blood pressure, but in most cases exciting causes may be demonstrated, such as severe exertion, straining on defecation, bloating, excitement during mating, during an operation, etc. As these exciting causes become active much more frequently in borses, heart rupture is observed more frequently in the horse than in other animals, with the exception of traumatic rupture of the heart in cattle.

Anatomical Changes. The pericardium contains more or less fresh blood (hemopericardium). A solution of continuity in the fibers of the heart muscle is often found in the walls of the ventricles, less often in the walls of the auricles. The tear is almost always small, its borders ragged and its path through the ventricular wall usually corresponds to a zigzag line, so that it is only with difficulty possible to introduce a probe into the ventricle. Sometimes the tear takes place in the interventricular septum (Dieckerhoff), and in such cases hemorrhage does not take place into the pericardium.

Symptoms. In general only symptoms of cardiac paralysis or of internal hemorrhage are evident, and these cause death rapidly because the heart is compressed by the blood or hindered in its diastolic dilatation, or then the action of shock upon the heart becomes manifest. The occurrence of a rupture is usually announced by sudden signs of indisposition. In a part of the cases death occurs suddenly, at night, or so rapidly that hardly any symptoms are ascertained aside from collapse and convulsions.

Treatment. A rupture of the heart which has actually occurred cannot be treated. Its appearance, however, may sometimes be prevented by removing the exciting causes.

10. Tumors in the Heart. Neoplasmata Cordis.

Tumors in the wall of the heart are of only slight clinical importance. Fibroma, fibromyxoma, myxoma, lipoma, sarcoma (in horses

melanosarcoma) and fibrosarcoma, also metastatic adenoma, carcinoma, exceptionally lymphosarcoma, angioma and lymphangioma occur principally in the walls of the ventricles. Fibrosarcomas are the most frequent growths, and in horses the melanosarcomas. Growths protruding into the heart cavities or into the pericardial sac not infrequently appear pedunculated.

The morbid symptoms are very uncertain in character. In most cases sudden death was observed, or the swellings were found on autopsy of such animals which had not presented any symptoms of cardiac disease during life. Only rarely disturbances of the heart function were noticed; in horses dyspnea was often diagnosed. Rautmann reported a case in a cow in which the right auricle was completely filled by a round-cell sarcoma and greatly dilated. The cow suffered from severe attacks of vertigo for six months, appeared emaciated and gave but little milk. It is probably only in exceptional cases possible to refer disturbances in the cardiac function to the presence of a swelling in the heart wall with any degree of certainty, especially when the occurrence of a primary new-formation in the organs that are susceptible to immediate investigation permit the assumption of a metastatic swelling in the heart.

Of animal parasites cysticerci and echinococci occur most frequently in the heart muscle or under the epicardium (Neumann, Rev. Vét., 1905, 729, with literature). The cysticerci do not appear to produce any noticeable disturbances in the heart function, and echinococci are found, not at all rarely, in the heart muscle of apparently healthy cattle, as is proved by the findings in abattoirs. (In exceptional cases the echinococci occur also in horses, especially in the heart muscle or in the wall of the aorta.) If the echinococcus bladder is comparatively large, or if several of them are present at the same time (Morot found 20 in one case) they naturally interfere with the heart action, but the cause of the disturbance cannot be determined even approximately. In a number of cases death occurs suddenly from paralysis or rupture of the heart, or from rupture of the echinococcus bladder into the heart cavity and the transportation of its contents into the pulmonary artery (Eggeling).

Of other parasites the Sarcosporidia occur in the heart muscle of sheep without leading to any particular cardiac disturbances; also mature specimens of Filaria immitis in the right auricle and the Strongylus vasorum in the heart of dogs (see page 599.) Cocu found a living specimen of distoma hepaticum in the coagulum attached to the chordae tendineae of the right ventricle of a cow dead with symptoms of severe cachexia and dyspnea.

12. Acute Endocarditis. Endocarditis Acuta.

Occurrence. Acute endocarditis belongs to the less frequent diseases and occurs in horses usually in consequence of influenza or of pyemic affections; in cattle it develops most frequently in the course of pyemia, of articular rheumatism, rarely of tuberculosis. Hogs are usually affected by an erysipelatous endocarditis; less often the affection accompanies swine plague, and has only in exceptional cases another origin (Lüpke, Bang,

author's case). In dogs the disease usually follows upon pyemia, septicemia, distemper, exceptionally also upon tuberculosis (Jensen observed 13 cases of ulcerating endocarditis among 3,240 sick dogs). In cats and birds acute endocarditis appears to occur only very rarely.

According to Bollinger and Zuern, a contagious, ulcerous endocarditis occurs in chickens, as a rule, only sporadically but sometimes also enzootically. Fox observed an enzootic of acute verrucose endocarditis in opossums.

Etiology. Acute endocarditis develops as a rule through an infection, but the various infectious substances concerned are at present only partially known. Comparatively often the pyogenic bacteria (cocci, streptococci, staphylococci, bacillus pyogenes) appear to be concerned in the production of the affection (Trasbot, Cadéac, Albrecht, Csokor, Nocard, Kitt, Bang, Lüpke, Fröhner, Joest, author's case). In hogs the erysipelas bacilli are usually of importance as etiological factors. In ulcerous endocarditis Jensen and Thomassen have found the bacillus coli communis in association with other bacteria; Kitt several times the botryomyces in horses; Luginger a streptothrix in cattle. The tubercle bacillus was found by Cadéac, Cadiot and Bergeon in cattle; the corynebacillus renalis by Cadéac and Scherzer in the deposits upon the affected heart valves; the bacillus bipolaris also is probably frequently a factor. For instance, Jensen found ovoid bacteria among other microorganisms in endocarditis of dogs, and Bang as well as de Jong observed endocarditis in swine plague (among 16,000 hogs dead of swine plague, 676 cases of endocarditis verrucosa were found in England). Acute endocarditis has also been noted in swine pest (de Jong).

Ettore describes several cases following an atypical septicemic form of blackleg with blackleg-like lesions in the myocardium in calves and yearlings.

In some cases an infectious endocarditis develops without an affection of the other organs and must then be considered as a primary disease. In the majority of cases, however, the trouble is secondary in nature and the bacteria reach the endocardium from inflamed organs (heart muscle, pericardium, mediastinum) by direct continuity or, much more frequently, from distant pathological foci with the blood stream. First in this respect are pyemic and septicemic diseases. Wherever in the body suppurating inflammations occur, like suppurating wounds, inflammation of the umbilical cord, variola (immediately before the suppurating stage or at its beginning), puerperal fever, strangles, fibrinous-purulent inflammation of the serous membranes, the malignant form of foot-and-mouth disease, etc., the opportunity for localization of pyogenic bacteria on the heart valves may be given. Sometimes seemingly quite inconsiderable losses in substance (ulcers at the tip of

the tail or elsewhere on the skin of dogs, bites, pressure wounds in horses, etc.) or inflammatory foci in other organs (in the case of Blanc's, inflammation of the bile ducts) afford a point of entrance to the pyogenic bacteria. The transmission of bacteria from the blood of the mother to the fetus is also possible, and this explanation must probably be accepted for cases that occur successively in several young of the same mother animal (Burke, Stephenson). Jensen and Fröhner even believe the transmissibility of the disease from animal to animal to be not impossible.

Contrary to the occurrence in man, acute endocarditis occurs only exceptionally in association with articular rheumatism in animals, but with greatest relative frequency in cattle. Rheumatic laminitis in horses at times causes an acute endocarditis, as do croupous and catarrhal pneumonia.

The bacilli of swine erysipelas are deposited in the tissue of the heart valves after a slight acute general infection, or as Jensen, Lohnsee and Eisenmann have shown, after the erysipelatous urticaria has run its course, and cause there an inflammation through which the apparently recovered animals perish in the course of a few weeks. Marek saw an acute endocarditis being produced by the erysipelas bacillus in two horses which had been employed for the production of erysipelas serum.

In many cases no bacteria are found in the territory of the inflammation, a fact that must be explained thus, that either the inflammatory virus had disappeared in the meanwhile, or that bacterial toxins absorbed from other organs had produced the disease.

Predisposing factors may be of import to such a degree that formerly many cases of endocarditis were referred to the immediate action of such factors. Among them are: cold (frequent in horses, according to Trasbot), over-exertion, traumatic influences upon the heart region, fracture of ribs, etc. The chronic form of endocarditis also predisposes to renewed acute inflammation (Fuchs).

Pathogenesis. The bacteria which circulate in the blood stream, probably rarely localize directly on the surfaces of the valves which are contiguous to the blood stream while this passes by them, less frequently on the parietal layer of the endocardium. Usually they are arrested in the capillaries of the valves, and also at the same time in the blood vessels of the heart muscle. The reason why the localization of the bacteria mentioned as the second contingency takes place also on that surface of the valves which is turned toward the lumen of the heart, lies, according to Eisenmann, in the peculiar arrangement of the blood vessels in the heart valves. The micro-organisms, which have localized, and their toxins cause an inflammatory condition in the tissue of the valves and in the

wall of the heart. The surface of the valves becomes rough and masses of thrombi are deposited. This results in a mechanical interference of the blood circulation, and the simultaneous myocarditis causes a diminution of the strength of the heart, in which the weakness of the heart is further augmented by a possible general infection, or by the circulating toxins of the virus.

Anatomical Changes. In most cases the valves of the left half heart are affected, less often those of the right half as well, or even exclusively (Endocarditis valvularis); the pulmonary valves are iuvolved in exceptional cases only. In this respect cattle make an exception in so far as in them disease of the tricuspid valves is more frequent. An inflammation of the endocardial lining of the muscular wall (Endocarditis parietalis) is rare.

In the verrucose valvular endocarditis the surfaces of the valves which face the blood stream or their borders disclose poppy-seed sized or somewhat larger nodules, which later frequently coalesce to form larger nodules, and become covered by coagula. In ulcerous valvular endocarditis ulcerations with reddened, uneven border and grayish-red or yellowish base are found, which contain considerable fibrinous deposit.

In consequence of the ulcerous destruction the valve becomes thinner, in some cases it is bnlged out by the blood pressure (Aneurysma valvulare acutum) and is eventually ruptured (A. valv. acut. ruptum). If the destructive process is rapid, irregular cleft-like openings form with swelled, gnawed borders (Valvula fenestrata) without aneurysmal changes. When the inflammation is continued to the endocardial lining of the heart, a purulent inflammation may arise in the heart muscle, giving rise to bulging and to rupture of the thinned muscular wall (Aneurysma cordis acutum). Occasionally the valve is even torn from the chordae tendineae or from its attachment.

Loose blood clots or loosened portions of the valvular tissue lead to embolism in various organs, which may be followed by purulent inflammation.

According to Egge, sharply-defined, turgid hemorrhagic vesicles the size of a pin-head to that of a pea, or suggilations of varying size at the free margin and at the point of attachment of the chordæ tendineæ of the bicuspid and tricuspid valves can be found in most calves during fetal life and up to three weeks old. They represent a physiological condition.

Symptoms. The clinical manifestations of acute endocarditis show great variance, depending on the nature of the inflammatory process, the causative agent, the different degrees of involvement of the heart muscle, the localization of the inflammation and on the presence or absence of a general infection. Generally the disease commences with a more or less

conspicuous affection, some animals *manifesting only* depression, dullness, slight rise in temperature, and tire easily, while others again show from the onset pronounced debility, complete inappetence and high fever. Between these two extremes there are numerous intermediary degrees of the affection.

The heart beat is usually increased or bounding and may produce a tremor of the chest wall which can be noticed from

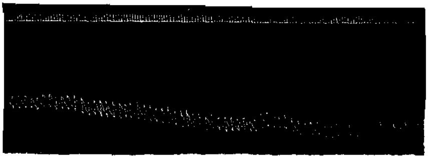

Fig. 157. Sphygmogram of the Art. femoralis in a dog with ulcerating endocarditis of the aortic valves. Pulse bounding and much accelerated. (Time indicated in 0.2".)

a distance. The rate of the heart beats varies; while it is increased by about one-half of the normal in light cases, it rises in severe attacks in large animals to 4 to 5 times, in small animals to 2 to 3 times the usual rate, and is frequently *arhythmic. The cardiac dullness is not altered at first, nor* in rapidly progressive cases through their entire course, but in more prolonged cases, in which the heart is dilated, the area of dullness is enlarged. The heart sounds may remain clear for a long time, indeed, to the end, except that they may become *somewhat feeble and dulled*, but in most cases they *eventually* become changed. Usually the two sounds run together into a single humming or roaring noise; at other times an endocardial murmur is heard instead of the first or second heart sound or in place of both, which is usually, soft, blowing or even has a certain resonance to it and is heard in varying intensity over different portions of the heart, just as in chronic endocarditis. If the inflammation occurs in several portions of the heart, several sounds may be perceptible which vary in pitch, intensity and volume.

The pulse is always accelerated, although its frequency is extremely variable in different cases; it is frequently arhythmical, irregular and intermittent; the intermissions are sometimes so frequent that the number of heart beats is as much as twice that of the pulse beats. At the commencement of light cases, the pulse is strong and even vigorous in relation to the rate, but later, and in severe cases from the first, it is weaker and soft. The character of the pulse depends in part upon the localization of the disease (see valvular diseases); for instance in inflammation of the aortic valves it is bounding (see Fig. 157).

In correspondence with the intensity of the disease circulatory disturbances develop which are manifested by cyanosis, fullness of the veins, negative venous pulse, and by frequent or difficult respiration, and which may be aggravated into asphyctic attacks on the slightest motion or through external influences, or they only become manifest in such attacks, while they are not present at rest. Another consequence of the circulatory disturbances, especially of a severe pulmonary hyperemia is in many cases a bloody discharge from the nose which may be accompanied by symptoms of pulmonary edema. Moreover, edemas sometimes develop quite early in various parts of the body, for instance, on the limbs and on the lower portions of the trunk. Occasionally, and particularly in hogs a decided weakness of the hindquarter, or even of one posterior extremity, is observed, which is due either to a stenosis of the aortic opening, or to a general embolic obstruction of the arteries, or to the debility which follows upon the severe general disease.

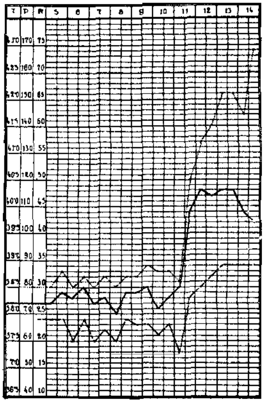

Fig. 158. **Temperature curve in acute endocarditis.** The disease had appeared in a Great Dane, brought to the hospital with an ulcer on the tail, and ended fatally in four days.

The temperature is raised in all cases (Figs. 158 and 159), but in slight attacks it is moderate, while severe cases are accompanied by high fever. There are, however, exceptions, and the great variability of the disease-causes makes it evident that the type of fever may be subject to numerous variations.

Disturbances in the general condition, which are always observed, may be so insignificant in cases running a slow course that they are not observed at all for a time, especially in swine which are more indolent and live in herds. In other cases the attack commences with considerable indisposition or debility; in horses symptoms of colic are now and then apparent.

In cases of ulcerons endocarditis, metastases may occur in various organs, producing purulent pneumonia, purulent nephritis, sometimes hematuria, acute meningoencephalitis, purulent inflammation of the joints, etc. The hemorrhages which appear in various parts may also be of similar origin.

With the gradual increase of circulatory disturbances death occurs in some of the cases, while in others, especially in those affected with the ulcerous form, symptoms of metastatic inflammation in various organs become evident. Such metastases may give rise to *purulent pneumonia*, or purulent nephritis sometimes accompanied by hematuria; to acute meningitis, purulent inflammation of the joints, etc. Similar causes must be attributed to the local hemorrhages which develop here and there, for instance, underneath the cornea.

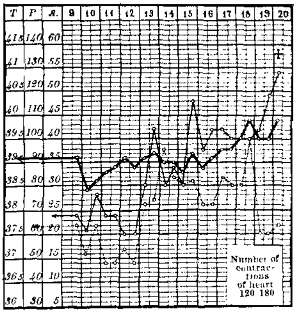

Fig. 159. *Temperature curve in endocarditis of erysipelas in a horse.* Two days before death the number of heart beats was twice that of the pulse beats.

In a very small percentage of cases a possibility of recovery may exist; but in animals which do not die the clinical picture of chronic endocarditis usually develops.

Diagnosis. The disease can only then be recognized positively when repeated examinations have demonstrated that valvular insufficiency or stenosis have developed in a previously *healthy heart in a comparatively short time.* A careful supervision of the heart action and repeated examinations of the heart are necessary the more, as the elimination of acute febrile diseases (anthrax, severe forms of influenza, septicemia, etc.), perhaps also of severe pulmonary diseases, is possible only in this manner. Of foremost importance is the character of the heart sounds and their transformation into murmurs, but even such findings do not afford absolute proof because similar phenomena may be observed in acute myocarditis and in cardiac dilatation. If the murmurs persist permanently, if to them are added circulatory disturbances and perhaps emboli, the nature of the disease may be assumed with sufficient certainty. Still it must not be forgotten that an animal ill with chronic endocarditis may become affected with another septicemic disease.

The differentiation of verrucous endocarditis from the ulcerous form is usually very difficult. In general it may be said that the more severe the symptoms, the higher the fever, and the more turbulent the circulatory disturbances, the greater is the probability of ulcerous endocarditis. Collateral factors

may afford information in so far as the verrucous form usually develops in connection with swine erysipelas or with articular rheumatism, while endocarditis ulcerosa occurs in purulent or ichorous processes anywhere in the body.

Prognosis. Acute endocarditis is always a severe disease, especially in the ulcerous form. Even in the lightest cases there is little hope for complete recovery, because usually chronic changes remain which afford a basis for permanent valvular lesions and thus reduce both the ability to work and the nutrition of the animals.

Treatment. Absolute rest should be procured first of all. For the alleviation of the excitement, which is noticeable at least occasionally, cold or ice compresses upon the heart or cold sprays are appropriate. If the heart action is very rapid and irregular the cautious administration of digitalis or of its preparations or of other cardiac remedies is indicated (see valvular diseases). In heart weakness the danger of cardiac paralysis must be counteracted with stimulants, for instance, with alcohol, wines, black coffee, tea, caffeine, theine, camphor, Liq. Ammon. Anisatus internally; ether, camphorated oil, tincture of musk subcutaneously. In excessively high temperature the fever requires lactophenin, antipyrin, aspirin, antifebrin, etc. Iu endocarditis of rheumatic origiu salicylic acid and its preparations may be tried.

13. Valvular Diseases. Vitia Cordis. Heart Troubles.

The designation of valvular lesions includes the chronic organic diseases which cause functional disturbances of the heart valves and the cardiac ostia. They differ in their derivation.

Occurrence. Since valvular lesions occur most frequently in consequence of chronic endocarditis which is most common in dogs, the valvular troubles are found most often in these animals. (According to Cadiot 5% of dogs over one year old suffer from valvular disease; among 793 dogs autopsied by Johne, chronic endocarditis was found in 4.3%, and among 1,831 dogs which were examined in the pathologic-anatomical institute in Budapest 3.9% were found with evidences of endocarditis). Next in frequency are horses, and then, very much less often, the other mammalians, if the cases of acute endocarditis are not included. According to Ball, cats are rarely affected with chronic endocarditis. The observations of Larcher and Hartl show that valvular diseases are not infrequent in fowls, although they give rise to functional heart disturbances comparatively rarely.

Etiology. Valvular heart lesions may be caused by the following diseases of the heart:

1. **Acute Endocarditis** (see p. 795), which in its less rapid cases takes a course presenting the clinical picture of valvular disease.

2. **Chronic Endocarditis** (Endocarditis valvularis chronica). This disease is the basis for most cases of valvular troubles and develops, probably principally, out of the acute inflammation; it either occurs thus that, after the acute endocarditis has run its course, a proliferation with subsequent atrophy of the connective tissue takes place, or thus that an acute inflammation, which is not very intense of itself, recurs periodically and produces proliferation and atrophy of connective tissue after each recurrence. Accordingly all causes and conditions which stand in etiological relation to acute endocarditis may occasionally also lead to a chronic inflammation. In this respect those factors especially enter into consideration which usually, or at least occasionally produce a mild acute inflammation. This is particularly the case in endocarditis following upon articular rheumatism and upon croupous pneumonia, also in inflammation due to tuberculosis and to erysipelas. In other cases the inflammation is chronic from the beginning and is usually not noticed; and in such cases an infection may be a causative factor, although it does not give rise to acute symptoms. Further, chronic endocarditis develops in connection with chronic endoarteritis and arteriosclerosis, and in dogs frequently in association with chronic interstitial nephritis. Sonnenberg found the mitral valve of a cachectic tuberculous cow thickened and covered with a yellowish-gray deposit, while the endocardium of the left heart showed diffuse grayish-yellow discoloration. Hartl found in chickens repeatedly a chronic endocarditis the origin of which could not be determined. Lastly, severe exertion or the mechanical irritation of the valves which is necessarily exerted by it upon them, and also cold are said to produce the disease. Coppel found hyperplastic changes on the atrio-ventricular valves with relative frequency in work-dogs.

3. Injuries of the valves from time to time play a part; for instance, when the valves become perforated, or torn from their attachments (semilunar valves), or from the chordae tendineae (tricuspid valves), during hard work or simply by the blood pressure. Exceptionally a simple overextension of the aortic valve may cause its inability to close (Zschokke).

4. **Tumors and Echinococci** may cause both insufficiency and stenosis if they grow from the annuli fibrosi or from the wall of a heart cavity toward an ostium. In examining a horse, Ball found a thrombus lying in both atrioventricular openings and preventing the closure of the valve.

Cholet found a subendocardial abscess the size of an orange in the region of the points of attachment of the chordæ tendineæ to the bicuspid valves in an ox.

5. **Diseases of the heart muscle** may produce a valvular insufficiency even if the valves are not injured (insufficientia relativa s. muscularis). Especially in the proper functioning of the auriculo-ventricular valves both the relaxation of the chordae tendineae and the contractions of the muscles around the ostium cooperate to a considerable degree. If the contractions are not sufficiently energetic, the closure of the valves becomes insufficient. A similar disturbance results if one of the ostia has been dilated excessively in case of cardiac dilatation of high degree. Insufficiencies from this cause are by no means infrequent and are usually due to chronic myocarditis, sometimes they follow upon a cardiac weakness from other causes and usually affect the right heart. Finally dilatation of the arterial trunks close to the heart may result in an insufficiency of the semilunar valves.

6. **Congenital heart lesions** must also be included here. According to Preisz they are usually due to impeded, less often to faulty development or to intrauterine heart diseases. A comparatively frequent anomaly is the persistence of an opening in the upper anterior portion of the interventricular septum (foramen interventriculare persistens) and according to Preisz the cases described by Csokor and Johne under the name of septum membranaceum persistens belong to this lesion.

The malformation may be accompanied by a dislocation of the septum as in the two cases in calves reported by Joest. The foramen ovale between the auricles remains open fairly often and also the ductus Botalli. It occurs only very rarely that only one auriculo-ventricular opening or a simple arterial trunk proceeding from the ventricle is found in addition to the above mentioned anomalies.

Anatomical Changes. Chronic endocarditis leads to new-formation followed by atrophy of the connective tissue; owing to these changes the endocardium is thickened in varying degrees, verrucous, polypoid or cauliflower-like excrescences are formed on the borders of the valve which is in contact with the blood stream, and the chordae tendineae are thickened. The thickened valves become rigid and the semilunar valves are transformed into thick flat pads, the separate portions of the bicuspid and tricuspid valves into thick, knobby formations; in exceptional cases contiguous parts of valves become adherent. The thickened chordae tendineae appear shortened. On the parietal layer of the endocardium changes are frequent but less striking and only rarely produce thickening. Jármai found in a horse that died from exhaustion, a chronic endocarditis with calcification of the wall and valves.

Severe changes of such character on the valves cause inability of closure of the valves, or constriction of the openings of the heart and not infrequently both. At the same time the parts of the heart lying above the stream flow disclose hyper-

trophy and dilatation. In the stage of insufficiency of the heart symptoms of blood stasis may also be present.

As regards the seat of the changes the disease affects in horses frequently the aortic valves (according to French authors this is the most frequent form of heart trouble in horses), more rarely the bicuspid valve. In cattle and goats the valves of the right heart are as a rule affected most frequently, in swine those on the left side of the heart, especially the bicuspid valve. In dogs mitral insufficiency is observed most often, then the simultaneous affection of bicuspid and tricuspid valves and about half as often disease of the tricuspid

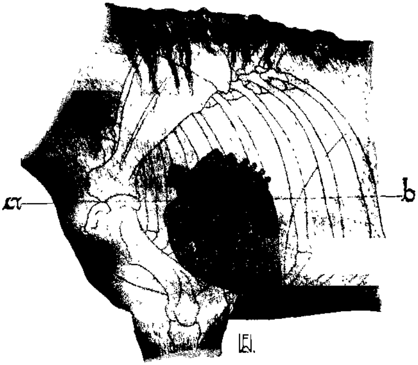

Fig. 160. *Location of the greatest intensity of endocardial sounds in the horse.* *a. b.* shoulder line; *1* punctum maximum of the murmur originating in the left auriculo-ventricular opening; *2* in the aortic opening; *3* in the opening of the pulmonary artery. In the other species similar conditions may be shown.

valve alone. According to Larcher and Hartl the valves of the right ventricle are usually affected in birds.

Symptoms. According to the localization of the morbid process the symptoms are so different that it appears proper to consider them separately.

(a) Insufficiency of the Aortic Valves.

In insufficiency of the semilunar valves of the aorta a slit-like opening persists between the valves during ventricular systole, in consequence of which a portion of the blood which had been forced into the aorta during the preceding systole flows back into the left ventricle, producing a murmur in so

doing. At the same time blood flows into the left ventricle from the left auricle, and the ventricle, receiving blood from two directions, is dilated, owing to its power of adaptation, but forces out its entire blood content during the next systole, for which a stronger contraction is necessarily required. The continuous increase in the activity of the heart gives rise to a hypertrophy of the wall of the dilated ventricle.

The symptoms of insufficiency of the aortic valves are as follows: The stronger contraction of the hypertrophied and at the same time dilated left ventricle produces an increased lifting heart beat on palpation of the cardiac region, especially after excitement, and a diastolic thrill of the wall of the thorax is noted on the left side. The area of heart dullness on the left side is displaced in dogs about 3-5 cm. or more upward, while in other animals it is displaced upwards and backwards and may reach as far as the seventh rib and beyond the middle of the lower third of the chest. The first heart sound is clear,

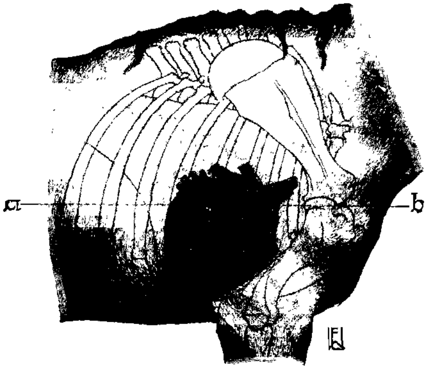

Fig. 161. Location of the greatest intensity of the murmur originating in the right auriculo-ventricular opening in the horse (1). The other murmurs the same as in Fig. 160.

but the diastolic sound is accompanied by a murmur which is usually loud, blowing or roaring, perhaps rough or ringing, prolonged, and which is separated from the systole by a rather short pause. Its greatest intensity is, in all mammals, in the left fourth intercostal space immediately below the shoulder line (Fig. 160.2), although it may be heard, a little less clearly, also in the same intercostal space on the right side. The purely diastolic sound which originates in the Art. pulmonalis can be heard in the region of the left ventricle in association with the murmurs just referred to, but near the origin of the

pulmonary artery (left third intercostal space, lower half of the lower third of the chest [Fig. 160.3]) it may be heard almost alone.

The pulse is characteristic (Fig. 162a). The hypertrophied left ventricle forces an increased amount of blood into the aorta

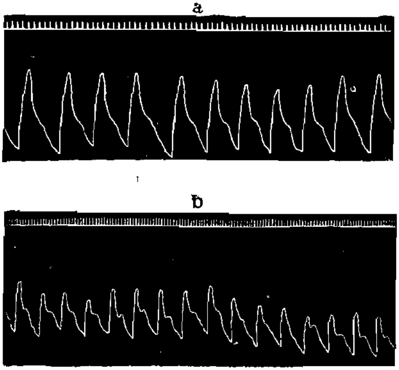

Fig. 162. Sphygmogram of the abdominal aorta in the horse. a, Bounding pulse in aortic insufficiency; b, pulse of a healthy horse (Time in 0.2").

and through it into the peripheral arteries in which the return flow of the blood during the preceding diastole had produced a very considerable lowering of the blood pressure in comparison with the normal. The increased amount of blood which is forced out with every ventricular systole, and the greater difference (compared with the normal) between the systolic and the diastolic blood pressure in the arteries produce a more vigorous and more rapidly increasing wave which disappears just as rapidly, because after the termination of the systole the blood can flow off rapidly toward the periphery and the heart. Accordingly the pulse is strong, large, rapid and bounding (Fig. 162a). A bounding pulse may be felt even in the smaller arteries, and indeed in the fundus of the eye the pulsation of the arteries can usually be seen clearly. The larger, and often also the smaller arteries show systolic pulsation and sometimes a systolic pulsation of the liver may be observed. At the same time a clapping vascular sound may often be heard even over smaller arteries. Finally the mucous membranes may suddenly become reddened synchronously with each ventricular systole; this redness may be emphasized by producing previously a circumscribed pallor by the pressure of a finger tip. This capillary

pulsation sometimes occurs in the severe cases of aortic insufficiency.

(b) Stenosis of the Aortic Opening.

In stenosis of the aortic opening the left ventricle is obliged to force the normal amount of blood through an opening which is smaller than normal, and this leads to the development of a systolic murmur and also requires an increased expenditure of energy which in its turn causes an increase in the thickness of the left ventricular wall.

The heart beat may therefore be heard over a larger territory and either more strongly or less strongly, because the so-called closure time of the heart function is omitted. Simultaneously with the heart beat a systolic thrill may usually be felt. The cardiac dullness is increased only inconsiderably. The first heart sound is covered by a very strong, prolonged systolic murmur, which is separated from the diastolic sound by only a brief pause; this murmur may be so strong that it can be heard in the neighborhood of the animal. Its maximal point is in the fourth intercostal space immediately below the shoulder line (Fig. 160.2). The systolic sound which originated in the right heart, as well as the transmitted murmur are heard on the right side also. The weakened aortic sound may be heard in the left fourth intercostal space, below the shoulder line, while the pulmonal sound is heard in fairly normal intensity in the third intercostal space in the lower half of the left lower third. The pulse wave is sluggish; its growth may be followed with the palpating finger, because owing to the aortic stenosis the systolic pressure of the arteries increases much more slowly than is the case under normal conditions. At the same time the pulse is remarkably small and presents a distinct contrast to the heart beat which is usually increased. The systolic murmur is transmitted even into the smaller arteries, for instance, those of the head and of the extremities.

(c) Insufficiency of the Bicuspid or Mitral Valve.

If the two flaps of the expanded bicuspid valve do not touch, a part of the blood in the left ventricle flows back into the left auricle at each ventricular systole, and the blood of the pulmonary veins is emptied into this auricle at the same time. This causes the pressure in the auricle to be increased, it becomes dilated and eventually hypertrophied, although the hypertrophy is usually not very marked. But since the auricular wall cannot expend the force sufficient to remove the increased pressure and since at each auricular systole blood is forced into the pulmonary veins which are not provided with

valves, the stasis is continued upward into the pulmonary veins and from these through the comparatively wide pulmonary capillaries, to the pulmonary artery and the right ventricle, the muscular wall of which is hypertrophied in consequence. There occurs therefore an increase in blood pressure affecting the entire lesser circulation, which is compensated in the right ventricle. The persistent passive hyperemia of the lungs gradually leads to the development of the socalled brown induration of the lung tissue. The muscle of the left ventricle also is usually thickened, although not greatly, because at each systole it is obliged to force out a larger amount of blood.

The heart beat is usually normal on the left side, but increased on the right side; in horses a contraction is observed commencing immediately after the beginning of the ventricular systole, instead of the normal well-marked systolic rise in the region of the heart during systole. Simultaneously with the heart beat a systolic thrill may often be felt. The cardiac dullness is not infrequently unchanged, but at times it is somewhat enlarged on the right side, which may be seen by the fact that in the fourth and also in the fifth intercostal space on the right side a distinct cardiac dullness is manifest. On the left side the cardiac dullness is only rarely noticeably increased. Instead of the first heart sound a prolonged, blowing or rushing systolic murmur is heard on the left side, the *punctum maximum* of which is in the fifth intercostal space, in the middle of the left lower third of the chest (Fig. 160.1). In the intercostal spaces lying in front of it the systolic sound originating in the right ventricle may also be heard more clearly, and it is audible quite distinctly in the right 3d-4th intercostal spaces (Fig. 161.1), the transmitted and much attenuated murmur being also perceptible. The diastolic sound is clear, the second aortic sound usually normal in strength, the second pulmonic sound (in the left third intercostal space in the lower half of the lower third of the chest [Fig 160.3]) much increased, clear and short, i. e., accentuated, because the blood column, which within the pulmonary artery is under an increased pressure, is thrown with greater force against the semilunar valves. The pulse presents no particular deviations, becoming weaker only in severe cases. The transmitted systolic murmur may usually be heard also over the larger arteries (carotis, first part of the abdominal aorta). In a horse with compensated mitral insufficiency, Noerr has made an electrocardiogram with the poles placed at the right anterior breast and the apices and has thus found a characteristic high and wide auricular oscillation.

Owing to the continued increased blood pressure in the lungs superficial and accelerated breathing is noted, and also bronchial catarrh. The patients are inclined to pulmonary edema.

(d) Stenosis of the Left Auriculo-Ventricular Opening.

In a narrowing of the left auriculo-ventricular opening the entrance of the blood from the auricle into the ventricle during the ventricular diastole is impeded, so that a dilatation of the *left ventricle is produced* with subsequent hypertrophy. But as in spite of the greater work accomplished by the thin-walled auricle it is not capable of preventing the damming up of the blood, the stasis is continued to the right ventricle through the pulmonary veins and arteries. This leads in its turn to an increase of pressure in the pulmonary circulation and to hypertrophy of the right ventricle, while the left ventricle rather becomes smaller.

The heart beat and cardiac dullness are the same as in the preceding lesions. Of the heart sounds the first one is clear everywhere, but the second is accompanied by a diastolic murmur which is most marked in the left fifth intercostal space, in the middle of the lower third of the chest (Fig. 160.1). The murmur is not usually loud and either is heard through the entire diastole, being separated from the systole only by a short pause, or only at the commencement, eventually only near the end of the diastole (presystolic murmur). Aside from the murmur the second aortic sound may be, and the second pulmonic sound always is, heard distinctly, the latter being even more accentuated than in mitral insufficiency. The pulse is always small and soft because the left ventricle forces less blood into the aorta. The lungs show the same disturbances as in mitral insufficiency.

(e) Insufficiency of the Pulmonary Valves.

The right ventricle in such cases takes up during each diastole of the ventricle double quantities of blood, whereby its accommodative dilatation causes a subsequent hypertrophy, with a gradual reduction of the driving power. The ventricle under certain conditions may dilate to such an extent that a relative insufficiency of the tricuspid valve results. The insufficiency is not followed by an after effect on the lungs and right heart as long as they are compensated by the increased amount of work of the right ventricle.

The heart beat and the cardiac dullness show similar conditions as in diseases of the left venous ostium. The first heart sound is clear everywhere; instead of the second sound a prolonged diastolic murmur which gradually becomes weaker in all directions is heard over the origin of the pulmonary artery (Fig. 160.3), while the second aortic sound becomes constantly more distinct. No particular changes are noted in the pulse. The disease is always accompanied by respiratory disturbances.

(f) Stenosis of the Opening of the Pulmonary Artery.

The blood which is forced out of the right ventricle against the pulmonary artery meets, in the diminished opening of the

pulmonary artery, an impediment which increases the work of the right ventricle and leads to hypertrophy of its muscle. The blood pressure in the lungs is lowered permanently, and the amount of blood in the pulmonary arteries becomes less as soon as the energy of the right ventricle diminishes.

The heart beat and the cardiac dullness show the same deviations as in the preceding form. Over the origin of the pulmonary artery (Fig. 160.3) a loud, prolonged systolic murmur is heard, and more posteriorly also the first heart sound which originates in the left ventricle; the second sound is clear but weakened over the pulmonary artery. The pulse is small and feeble. Respiratory disturbances and cyanosis are noted from the first.

(g) Insufficiency of the Tricuspid Valve.

If the unfolded tricuspid valve does not close completely, a part of the blood in the right ventricle flows, during systole, back *into the right auricle through a more or less narrow opening*, the auricle is dilated and eventually slightly hypertrophied.

Since the auricular wall is capable of exerting only a slight force, and since the openings of the venae cavae are not closed by valves, the stasis of the blood is continued into venae cavae and into the other veins of the body.

The right auricular wall soon becomes exhausted and its cavity permanently filled with blood. By each ventricular systole, the blood driven back into the auricle as well as into the vena cava communicating with it, creates a sudden rise in pressure which is *continued up the jugular vein and the other large* veins, producing in them a ventricular systolic pulsation. This at the same time greatly increases the stasis already present before the exhaustion of the wall of the auricle took place.

The heart beat differs from the normal at most in so far as a systolic thrill is often felt simultaneously in the 3d-4th intercostal spaces. The cardiac dullness is not altered. The first heart sound is heard in the left 5th-6th intercostal spaces, either quite clear or together with a very faint transmitted murmur; the systolic murmur reaches its greatest intensity in the right 3d-4th intercostal spaces toward the middle of the lower third of the chest. (Fig. 161.1). The diastolic sound is either of normal intensity or frequently becomes weaker and in such cases the pulse becomes feeble and small. The veins are filled strongly; the jugular veins appear as thick cords and show a systolic (positive) pulsation following immediately upon the heart beat, which may, however, not be perceived owing to a long hair coat, especially in dogs. If the auricle could not remove its entire blood content, owing to its rapidly diminished functioning power a certain amount of blood is forced at each systole from

the right ventricle into the right auricle and from this into the vena cava. By the regurgitation of the blood into the venae cavae a systolic wave is produced and is continued to the jugular veins. The systolic venous pulse (Fig. 163) represents a pathognomonic phenomenon of tricuspid insufficiency, but it must not be confounded with the presystolic (negative) pulsation, which also occurs in this heart lesion and precedes the heart beat, or with the pulsation of the carotis. Venous stasis leads soon to edemas.

Fig. 163. *Cardio- and phlebigram of a horse with insufficiency of the bicuspid and tricuspid valves.* *A* Cardiogram. Instead of the elevation which occurs normally at the beginning of ventricular systole, a descending line is noted (*a—c*), due to the omission of the closure of the right ventricle; the line is interrupted by a small apex (*b*) which corresponds to the systole of the left ventricle. At the beginning of ventricular diastole (*c—d*) the pressure in the ventricles suddenly increases. *B* Phlebigram. α negative venous pulse; β positive venous pulse; γ carotid pulse; δε rapid emptying of the jugular vein at the beginning of the ventricular diastole.

(h) Stenosis of the Right Auriculo-Ventricular Opening.

A narrowing of the right auriculo-ventricular opening interferes with the flow of the blood from the right auricle into the ventricle, thus causing slight hypertrophy and at the same time a considerable dilatation of the right auricle. Owing to the latter cause blood-stasis occurs in the venae cavae from the beginning and is continued toward the periphery. On the other hand the other portions of the heart receive less blood and therefore become smaller. In consequence of the diminished pressure in the right ventricle and in the pulmonary artery thrombi easily form in them which may lead to embolism in the blood vessels of the lungs.

The heart beat is weak, a cardiac dullness can either not be demonstrated at all or it is diminished in extent. The first heart sound is clear, the second is accompanied by a presystolic or diastolic murmur (for similar reasons as in mitral stenosis) which is heard most strongly in the right 3d-4th intercostal spaces in the middle of the lower third of the chest (Fig. 161.1). The pulse is feeble and small. The veins are filled strongly and

a presystolic (negative) venous pulsation, as well as undulation, may be observed. The characteristic signs of severe blood-stasis, especially marked dyspnea, possibly attacks of asphyxia, occur already early in the disease.

(i) Congenital Heart Lesions.

Clinical manifestations of congenital heart lesions have been described only rarely. The most decided symptom usually consists in considerable weakness. The newborn animals are not capable of looking for their mothers. They stand with hardly a movement or cannot even keep their feet. Sometimes at rest, more often on motion, severe dyspnea and cyanosis are observed. Congenital valvular lesions give rise to the same local symptoms as the acquired forms, but murmurs are mostly absent in cardiac lesions which are due to faulty development, or they appear only in those cases in which there is a difference in the pressure in two communicating heart portions, provided the abnormal opening is not too large. In two cattle with persistent interventricular openings Huynen observed a systolic murmur in the left heart region and an accentuated diastolic heart sound.

The newborn animals usually die after a short time or are killed, as they are not fit to be raised. In rare cases they develop normally and may even live a fairly long time.

(j) Combined Valvular Lesions.

The most frequent combinations are those in which a stenosis of the related ostium coexists with insufficiency of a certain valve. Since valvular insufficiency is, in a way, the opposite of stenosis, the individual components of the clinical picture lessen each other; an exception occurring only in the accentuation of the second pulmonic sound in combined disease of the left auriculo-ventricular opening. On this account the discernible deviations lose more or less in distinctness and are often confused into a clinical picture which is difficult to analyze. In insufficiency of the aortic valves and coexisting aortic stenosis, for instance, systolic as well as diastolic murmurs are discovered, while the pulse is either not bounding or only slightly so, because the variations in pressure in the arteries are not so considerable and less abrupt.

The clinical picture is even more complicated if two or several ostia and valves are involved simultaneously. Frequently only portions of one half of the heart are diseased, for instance, the aortic valves and at the same time the bicuspid valve, but sometimes morbid changes are found in the right as well as in the left halves of the heart, as in diseases of the left venous or arterial ostium simultaneously with tricuspid insufficiency.

As it is possible for two anomalies to occur in each of four places, i. e., insufficiency and stenosis, and as these eight individual forces may enter the most variable combinations with

each other (a total of 247 combinations is possible), the variability of valvular lesions observed in the different cases is not astonishing, and at the same time renders descriptions of the clinical pictures suitable for all possible contingencies an impossibility.

Insufficiency of a valve or stenosis of an ostium disturbs the normal process of the circulation of the blood by producing a permanent rise in the blood pressure in the parts of the heart which lie proximally to the seat of the disease, and, in diseases of the left venous ostium, also in the pulmonary vessels. In order to maintain a normal, or at least approximately normal velocity of the blood current in the body and a normal blood pressure, certain parts of the heart are forced to accomplish continuously an increased amount of work. This causes their walls eventually to hypertrophy, and the heart is then enabled to accomplish the plus of work without constantly drawing upon its reserve strength. As long as the increased work of certain parts of the heart maintains the normal velocity of the blood current, and as long as therefore the functions of the various organs rest undisturbed in spite of the existing valvular lesions, with the exception of certain local disturbances which are constant accompaniments of some heart lesions: a compensation of the valvular lesion is said to exist. However, in this stage the function of the organs is usually undisturbed only in a condition of rest, in greater muscular exertions and still more in over-exertion transitory signs of incompensation may appear. Sometimes, especially in insufficiency of the aortic valves, the heart may be equal even to these excessively increased demands. On the other hand, in certain forms of valvular troubles the function of the organs is impaired to a certain degree, even while at rest, in spite of existing compensation; for instance, in diseases of the left auriculo-ventricular opening, the increased pressure in the lungs causes superficial and hastened respiration, while in aortic stenosis symptoms of cerebral anemia may exist.

The anatomical changes afford a satisfactory explanation why in diseases of the aortic ostium the symptoms of stasis generally become manifest later than in other valvular diseases. Not only is the most vigorous portion of the heart back of the affected portion in this case, but the weakening of the left ventricle causes a notable stasis only in the lesser circulation, which can moreover be compensated by the right ventricle. If, on the other hand, the right ventricle is obliged to compensate a heart lesion, the stasis continues, immediately to the right auricle and to the whole venous system, when the right ventricle becomes fatigued. Most unfavorable are conditions in diseases of the right venous ostium because the thin-walled right auricle is hardly capable of producing a compensation worth mentioning; in valvular diseases of the last variety a stage of compensation can therefore hardly be considered.

The cause of the permanent incompensation or decompensation which develops after a certain time can at present not be explained satisfactorily for all cases. In a number of cases the fact that the heart muscle is finally incapable of overcoming the constantly increasing difficulties must probably be attributed to the gradually increasing obstacles to the blood current. A similar effect results from frequently repeated severe muscular exertion, and from pulmonary edema which is not rare, especially in horses. In other cases the heart muscle is weakened by myocardial inflammation which developed simultaneously with or in consequence of the endocarditis. The strength of the heart muscle is also reduced by an extensive degeneration, while a slight degree of degeneration is not important in this respect. Finally, a functional disturbance of the heart muscle must be assumed, in a considerable number of cases, to form the basis of incompensation, which cannot at present be demonstrated histologically.

The incompensation is manifested by evidences of heart weakness.

Loosened portions of thrombi finally may give rise to embolism in various organs. In this manner emboli may get into the lungs from the right heart and into the other organs from the left lung. Cerebral emboli and also cerebral hemorrhages may be followed immediately by sudden death.

Course. Valvular disease is characterized by a very changeable course. Aside from the fact that its influence upon the circulation varies according to localization and intensity, the course is modified by external conditions and by work. While dogs which often pass their lives in quiet comfort not rarely live for years with valvular disease in animals which are employed for work, especially in horses, this becomes aggravated, relatively early, to such a degree that they become useless for exertion. For the same reason symptoms of stasis, the development of which requires considerable time, are observed in horses more rarely; they show instead signs of rapidly increasing dyspnea and usually die suddenly of pulmonary edema, cardiac paralysis or rupture of the heart, during work or while in motion. In dogs the gradual development of the sequelae of cardiac anomalies, up to severe edematous infiltrations, can be observed far better; in cattle and in hogs there is far less opportunity because these animals are usually slaughtered before the occurrence of these symptoms.

The disease takes a more rapid course in very young and in very old animals, because both lack the strength which is required for a sufficient compensation. Congenital heart lesions lead to death, with symptoms of severe blood stasis, usually during the first few weeks or months of life, although occa-

sionally the animals become older. Such cases have been observed by Thomas, Walley, Johne, Hutyra, Liénaux.

Diagnosis. The recognition of valvular disease is only possible on the basis of an exact examination, which also considers the clinical pictures described above.

Sometimes so-called anorganic murmurs are heard without valvular lesions and without any impediments to the circulation in the heart; these murmurs are heard more frequently during systole, only very rarely during diastole. These murmurs may be eliminated if in addition enlargement of a portion of the heart or accentuation of the second sound can be demonstrated, or if a thrill can be felt (*"frémissement cataire"*).

The absence of murmurs does not exclude the presence of a valvular lesion, because in case of slight insufficiency or stenosis, especially at rest, murmurs are not produced which are transmitted to the chest wall with sufficient intensity, and because in severe insufficiency the formation of murmurs may not occur, as in such a case the regurgitation of the blood does not take place through a narrow slit; finally, in the stage of incompensation too feeble contractions of the heart may cause the murmurs to disappear. In all these cases cardiac remedies and muscular exertion which have the effect of strengthening the contractions of the heart usually cause the murmurs to reappear. It appears therefore advisable in all cases in which a suspicion of heart disease exists, which includes all cases of dyspnea the nature of which is not quite evident, to examine the animals immediately after work and particularly to auscultate the heart region. Nor should it be forgotten that in aortic aneurysm murmurs are heard which correspond to the endocardial murmurs.

In domestic animals the recognition of the several valvular anomalies is always successful in the stage of compensation, sometimes also in compensated cases, if the character of heart beat, heart sounds and cardiac dullness is observed and the punctum maximum of the murmurs is ascertained.

The nature of the pulse is of great importance for diagnosis; in fact, its significance is often decisive, and the same is true for the character of the arteries and veins. Finally the frequency of the several valvular anomalies must be considered on making a diagnosis.

With the determination of a valvular lesion the proof has not yet been afforded that this lesion is based upon a chronic endocarditis, although valvular disease depends upon this in a great majority of cases. The actual cause of a heart lesion can only then be ascertained with absolute certainty in living animals if the development of the disease could be traced from an acute endocarditis, in connection with an infectious disease or with chronic arthritis. On the other hand, the absolute disappearance of endocardial murmurs and of the symptoms of insufficiency, after the systematic administration of cardiotonic

remedies and during physical rest admits the assumption of a muscular insufficiency.

Prognosis. Although the prognosis of chronic valvular lesions is always unfavorable in regard to the possibility of healing, the various forms of valvular anomaly require different interpretation with respect to their effect upon life and the usefulness of the animals. Stenosis of the ostia is in general a more severe disease than is insufficiency of the related valves, and disease of both valves of the heart is always more dangerous. Of comparatively the least dangerous significance are diseases of the aortic opening, because the strong-walled left ventricle maintains its functioning power for a fairly long time. Disease of the left auriculo-ventricular opening must be judged less favorably because the right ventricle is capable of less exertion and because in addition oxidation is interfered with by the persistently increased pressure in the lungs. The valvular lesions which are localized in the right heart are always of very unfavorable significance; that of the pulmonary opening because the lungs receive less blood, that of the right auriculo-ventricular opening because the auricle which is to produce the compensation is incapable of doing so.

The symptoms of incompensation are always serious, and the more so the less they are to be referred to external conditions. Of course, the prognosis is the less favorable the less the animal can be protected against exciting and exhausting influences. If the disease has existed only a short time, the age and general condition of the patient must be considered.

Treatment. Since healing of valvular lesions is impossible of accomplishment, the treatment must attempt to postpone an aggravation of the fairly good condition by a proper regulation of the diet, as long as symptoms of incompensation have not made their appearance. The patients must therefore be protected from all influences which impose an increased amount of work on the heart, which suddenly change the blood pressure or irritate the heart in any way. It is therefore necessary to regulate the diet thus, that the animals receive no irritating substances with their food (which must be insisted upon especially in carnivora), that not too much food is taken in at one time, but that the usual daily ration is eaten in several small portions. They must be supplied with pure air or led around in the open. The work done by the patients must be supervised very carefully. Experience has shown that, if these precautions are observed, animals with heart disease may do satisfactory work even for years, especially in agricultural employment.

Finally the digestion must be watched because fecal retention increases the abdominal pressure, and therefore the activity of the heart. In such cases the administration of mild laxatives,

especially of mineral salts, castor oil, rhubarb or aloes is indicated.

In the stages of incompensation (and veterinary aid is usually requested only at this time) absolute rest must be insured and the action of the heart regulated. Above all digitalis does good service because the heart contracts more vigorously and less frequently under its influence, so that the muscles obtain more rest during the longer pauses. Larger animals should receive, if possible, powdered fresh leaves (horses 2-4 gm., cattle 4-6.0 gm. daily), dogs an infusion (1-2.0 gm. in 200.0 gm. of water; 2-3 tablespoonfuls daily), and the nitrate or acetate of potassium may be added to increase the diuresis. According to the investigations of Kunz-Krause & Röder it appears better to employ folia digitalis dialysata (for larger animals 5-15 cc. per os) or the digitalysatum Bürger (20-30 drops subcutaneously for smaller animals). Maceration of digitalis leaves (maceration in pure water for 12-24 hours) may also be given to advantage. Digalen has an excellent action and is to be recommended, especially if dangerous cardiac weakness is present (horses 5-15 cc., cattle 20 cc., subcutaneously; or 15-30 cc. intravenously; dogs 5-15 cc. or 0.857 cc. per kg. of body weight), but if required for prolonged treatment it will be employed only in valuable or valued animals. Digitalis being rendered inactive in the stomach of the ruminants (Salvisberg), those preparations should be selected for these animals which are adapted for subcutaneous administration. Diehl observed abortion in pregnant cows after the use of digitalis. The tincture of strophanthus (10-25.0 gm. for large animals, the same number of drops in smaller animals, daily) has been found unreliable in contrast to strophanthinum depuratum, Merck, the good action of which is constant. It is given subcutaneously in aqueous solution (soluble in 40 parts of water) (0.002-0.003 gm. for horses, 0.00025-0.001 for dogs). At the point of injection painful swellings occur and sometimes necrosis of the skin (Fröhner, Author's case). Caffeine also has an excellent effect (caffeine sodium-benzoate or sodium-salicylate 6-8.0 gm. for large animals; 0.5-1.0 for small animals subcutaneously).

In pulmonary stasis, and also in general, moderate venesection may prove advantageous.

In case of distressing palpitation cold compresses upon the region of the heart will be found beneficial, also subcutaneous injections of morphine (0.20-0.50 gm. or 0.01-0.03 gm.). Liénaux treats the annoying cough of coexisting bronchial catarrh successfully with apomorphine (0.005-0.05 for large, 0.003-0.005 gm. for smaller dogs, subcutaneously).

In edema of the subcutaneous connective tissue calomel may be tried, since in human medicine a remarkable increase in urine and a rapid diminution of the edemas have repeatedly been

observed after its administration (for dogs 0.05-0.10 gm. 3-4 times daily alone or with an equal amount of jalap. The other diuretic remedies (acetate of potassium, squills, etc.) may also be employed and at the same time the daily amount of drinking water must be reduced. Agurin is very effective (for dogs daily 1-3 gm. dissolved in water), also theocin (in like doses), and theophyllin (in like doses). Together with the diminution of the subcutaneous edemas the transudations in the body cavities frequently also become less, and these may, moreover, be removed temporarily *by repeated puncture. If this is followed* immediately by a reaccumulation of serum, it should not be repeated.

Finally an existing anemia is treated by the systematic administration of iron and by appropriate feeding.

SECTION III.

DISEASES OF THE BLOOD VESSELS.

1. Chronic Induration of the Arterial Wall. Endoarteritis Chronica.

(*Endoarteritis chronica deformans. Atheromatosis, Arteriosclerosis, Atherosclerosis.*)

By the term arteriosclerosis is understood a chronic change in the arterial wall consisting of a decreased elasticity, increased consistency, proliferation of the connective tissue and frequently also a deposition of calcium salts. This is either a pure degeneration (atherosclerosis) or is due to the influence of inflammatory irritation (arteritis chronica). The lesions may occur extensively throughout the arterial system (generalized arteriosclerosis) or may be limited to small circumscribed foci especially in the region of the aorta (circumscribed, focal arteriosclerosis).

Occurrence. Also in animals, arteriosclerosis usually develops as a phenomenon of old age, but is not infrequently observed in young animals, where it develops especially upon an inflammatory basis. Parisot observed it in animals only a few months old. It occurs most frequently in herbivorous animals and is even found in wild animals (according to Paisot in hares, chamois, deer and wild boar). With few exceptions and in contrast to man, it is limited to the large arterial trunks.

Lüpke saw four cases of periarteritis nodosa in axis-deer, which affects almost all arteries of the general circulation, not excepting the smallest blood vessels.

According to an inquiry of M. Schmidt an endoarteritis of the aorta could be shown macroscopically in 0.5% out of 2,069 horses autopsied in the pathologic-anatomical institute in Budapest; according to Kitt a chronic inflammation is frequently met with at the origin of the aorta of older horses. Lyding found endoarteritic changes in the aortae of 35 out of 100 cattle, and out of 10 each of horses or dogs these changes could be ascertained in two each.

Etiology. Although the etiology is not known, it is undoubtedly of a multiple nature. In the forms originating through primary degeneration, the decrease in the efficiency of the arterial walls may be of pronounced significance. It is produced particularly by the action of toxins, which lead to a disturbance in the nutritive metabolism and the functional burdening of the arterial walls by increased blood pressure as a result

of bodily overexertion or under the influence of substances increasing the blood pressure. Of the toxins which may be brought in probable relationship to arteriosclerosis we may name in the first place bacterial toxins which exert their injurious effect in the course of the different infectious diseases. Thus, Lignières observed enzootic arteriosclerosis of the aorta in cattle and sheep in the Argentine Republic, which was due to an intestinal infection essentially the same as that described by Sivori as "La Mancha" and not to Bacillus pseudotuberculosis of Preisz-Nocard. Similar cases were also observed by Torrance. Other substances, particularly exogenic toxins as well as autotoxins, may take part in the production of arteriosclerotic lesions. In this connection physicians lay the blame upon the excessive use of alcohol and tobacco and upon lead poisoning. Regarding animals, autotoxins and the injurious waste products produced under the influence of very radical changes in nutrition probably play an important rôle.

Reports concerning the artificial production of arteriosclerosis in animals are quite numerous. Such lesions may be produced by the introduction of substances which, on the one hand, increase the blood pressure and on the other hand have a more toxic effect. These lesions consist of necrotic foci in the media with subsequent calcification, and slight changes in the intima; or, again, in fatty degeneration of the intima, hypertrophy and degeneration of the elastic fibers, and deposition of cholesterin and salts of lime in the intima and media. These lesions, which differ greatly from those found in arteriosclerosis in man, may, according to some authors, still be placed parallel with the latter, and, therefore, are also spoken of as true arteriosclerosis, since the difference, on the one hand, may be solely due to the difference in the anatomical structure of the arterial wall (absence of the elastico-muscular layer in the intima of cattle and rabbits), and, on the other hand, to the much more rapid development of the artificially produced lesions. Such lesions in the wall of the aorta have been particularly produced in rabbits by the intravenous injection of adrenalin continued for several weeks (Josué, Erb, Kuelbs, Fischer, Scheidemandel, Korányi, Ziegler and others), in which the toxic action of the adrenalin upon the arterial wall seems to be of significance. The lesions develop in spite of simultaneous injections of amyl nitrite, which lowers the blood pressure. Furthermore, Reinecke produced similar lesions with barium chloride, while Philosophow did likewise with salts of mercury, lead and zinc, although these substances do not increase the blood pressure. Particular significance must be attached to experiments of Saltykow with saprophytic as well as killed virulent streptococci in which the lesions of the wall of the aorta are said to be identical with those of true arteriosclerosis. More recently Saltykow attributes the arteriosclerosis thus produced to the action of the

cholesterin in the food, similarly as in the cases of arter sclerosis produced by him in rabbits by feeding milk and p tein. Also Wacker and Hueck assume the increased amount cholesterin in the blood as one of the causes of artificial arter sclerosis. This can be achieved either by the addition of chol terin to the food or by muscular exertion, dyspnea or by feed narcotic substances. In rabbits, such experiments lead to development of lesions, particularly in the intima, which very closely related to arteriosclerosis in man, especially if sm quantities of vegetable substances are added to the protein fo of animal origin. In dogs and rabbits, Loeb produced simi lesions when he fed lactic acid, and these lesions in dogs to their origin in the intima. He is, therefore, of the opinion th arteriosclerosis may also occur under natural conditions throu the toxic influence of lactic acid when it is produced in larg quantities, as during an insufficient supply of oxygen, increas bodily exercise, etc. According to Thoma, the deciding rôle, naturally occurring as well as in artificially produced arter sclerosis, is played by a permanent slowing down in periphery of the blood stream during a weakened condition the body, which causes a greater expansibility and increas tension of the wall of the blood vessels under the influence nutritive disturbances or over-expansion during excessively creased blood pressure. Particularly under such conditions, regular velocity in the periphery is said to be restored and generation of elastic and muscular fibers takes place, which turn reduces the pathologically increased tension in the injur walls of the blood vessels below the average and thereby brin about a structure of the intima consisting more or less of co nective tissue.

Circumscribed endoarteritis very frequently arises horses under the influence of the Sclerostomum vulgare, pref ably in the Arteria mesenterica anterior, not rarely in the ao or in the arteries of the extremities, exceptionally also in arteries of the other organs, including those of the hea (Cadiot, Piot-Bey, Magnin). A similar influence is exerted the much less frequent Spiroptera sanguinolenta upon the ao of the dog, the Filaria Poelsi (Vryburg) or the Filaria Bl (Carougeau & Marotel) upon the aorta of buffalo in Sumat sometimes also upon that of cattle, without, however, causi disturbances in the health of the animals.

Rautmann observed a case of tuberculous endocarditis with calcification a horse. Petit saw a case of tuberculous periaortitis in a dog with ulcerous foration into the lumen of the aorta. Raschke reported a case of generali arteriosclerosis in a cow with a medium degree of tuberculosis of the org According to the observations of Seibert, inflammatory induration of the arte in cattle may be exceptionally produced by foreign bodies or by a compressio the abdominal aorta by tumors. Roquet reported such a case in a horse du lymphosarcoma. Luepke could not determine the cause of a periarteritis no in axis-deer; neither could Guldner in a four-weeks-old calf and Joest and E in two hogs.

Anatomical Changes. In the incipient stage of the process small, elongated or round, white or pale yellow, shallow elevations are found on the intima of the artery, which are most numerous at those places at which the blood is thrown most strongly against the wall, for instance at the turns of main arteries, at places where branches are given off, etc.

In the case of Raschke, all arteries of the trunk, head, and extremities of a cow were highly changed and calcified, the arteries of the abdominal wall took a tortuous course, while the arterial branches of the abdominal viscera and the arteries of the lungs showed only slight changes. Also in the diseased basal arteries of the brain, the arteriosclerosis was but little developed. Corresponding to these spots there exists, in horses and cattle, a proliferation of the connective tissue, with a slight fatty degeneration and calcification, in dogs the media and intima are atrophied and there is fatty and calcareous degeneration. Cavity formation is rare, ulcerous destruction (atheromatous ulcer) is completely missing (Lyding). Eventually the arterial wall bulges and becomes rigid in consequence of diminished elasticity.

In periarteritis nodosa, the wall of the arteries show nodular thickening, hemorrhages, necrobiosis and constriction or total obliteration of the lumen (arteritis obliterans); at other points there is the formation of aneurysms with thrombi, while in hogs the lesions of the wall represent elongated knots (Joest and Hartz).

Concerning the induration of the aorta in the different species, one may conclude from the observations of Krause, that in cattle an extensive thickening of the intima with proliferation of connective tissne and neoformation of elastic fibers begins in the second year and progresses towards the heart, whereupon droplets of fat are deposited at the boundary between the media and thickened intima, which thickening rarely assumes a nodular character. Calcification of the aorta is a regular occurrence, beginning at about the third to fourth year. It begins in the abdominal aorta, which is changed only to a limited extent and frequently occurs without previous fatty degeneration of its tissues. Calcification of the aorta and a similar condition of the smaller arteries are reported by Lignières as phenomena of "Enteque" in Argentina. A similar condition is found in enzootic arteriosclerosis in sheep. Old goats may also suffer with arteriosclerosis (Schlegel, Krause).

In old horses (18 years or so) one finds arteriosclerosis which is less pronounced towards the heart. Sometimes it is nodular, similar to that found in cattle. The calcification of the media of the thoracic aorta, usually without previous degeneration, and which progresses centrifugally with age, but not as a rule extending to the abdominal aorta, is a regular lesion in old horses.

In dogs, beginning at about the fifth year, thickening of the intima with connective tissue proliferation, deposition of fat and early neoformation of elastic fibers take place at points where the calibre of the arteries is reduced. It regularly occurs in older dogs, while calcification is not a regular phenomenon of old age.

Calcification of the aorta is frequently found in old birds. Similar lesions may be produced in pigeons by Hypodectes columbarum.

In practically all young as well as in old dogs, Strauch found a probably physiological cross-striation of the femoral artery, as well as in the other main branches of the aorta, due to a contraction of the media or as a result of callous formation following rupture of the elastica interna.

Symptoms. A generalized arteriosclerosis can, in a fairly advanced stage be recognized only if the vessels which are immediately accessible to examination are involved. The walls of the subcutaneous arteriosclerotic vessels give to the touch a peculiar rigid and sometimes uneven firmness, the pulse is sluggish but at the same time fairly strong because the left ventricle is usually hypertrophied. The aortic sound is sometimes accentuated in consequence.

The changes in the vascular walls may occasionally be recognized in the abdominal aorta and in its pelvic branches. In other cases the presence of the disease can only be assumed when the left ventricle becomes hypertrophic without manifest cause or when symptoms of thrombosis or embolism make their appearance.

Periarteritis of axis-deer is, according to Lüpke, manifested in gradually increasing digestive disturbances which show much similarity with the clinical picture of chronic intestinal catarrh. In spite of fairly good appetite a gradually progressive emaciation becomes noticeable which passes into a sickly condition lasting for weeks and months.

Epizootic arteriosclerosis of the aorta in sheep causes dyspnoea, asphyxiating attacks, and sudden death, especially when the animals are urged to move rapidly; nutrition disturbances are in most cases absent.

For the symptoms of circumscribed arteriosclerosis the following chapters should be consulted.

Treatment. The further development of an established inflammation of the arteries cannot be arrested in any way and can not even be retarded. In human medicine iodide of potassium is administered internally in arteriosclerosis. Korányi succeeded in obtaining a favorable influence upon the process, which had been produced in rabbits by injections of adrenalin, or with subcutaneous injections of iodipin, but Fleischer & Loeb could not confirm his results.

2. Dilatation of the Aorta. Aneurysma Aortae.

Etiology. The walls of the aorta and those of the arteries

in general, with very few exceptions, become dilated only when their power of resistance has been reduced, for instance in the presence of chronic endoarteritis, especially when the blood pressure is lastingly raised. This evidently accounts for the fact that aortic aneurysm is observed most frequently in horses because these animals are often employed for hard labor.

According to Tombari the severe exertions which hunters are obliged to undergo may be considered to stand in causal relation to the frequency of aneurysm, and according to Palat the same holds true for 'bus horses.

In exceptional cases the commencement of the development of an aneurysm corresponds, as to time, with external influences, such as moving a heavy load, jumping, being thrown, or struck on the trunk, etc., or it may be referred to such an accident with a degree of probability. In such cases the traumatic factor of a sudden excessive rise in blood pressure might cause a partial tear in the aortic wall, in consequence of which the weakened arterial wall eventually bulges out. On the other hand it is not impossible that even in such cases the wall of the vessel was not quite healthy before the accident.

Anatomical Changes. A portion of the aorta is either evenly dilated (Aneurysma cylindricum) or the healthy portion gradually passes into a dilated segment, which again gradually passes into the healthy part of the vessel (A. fusiforme), or only a short segment of the vessel is sacculated (A. sacciforme). In the latter case the sacculation either affects the entire circumference of the vessel (A. sacciforme periphericum s. axiale) or only a part of it (A. sacciforme semiperiphericum). The sacculated bulging may reach in horses the size of a man's head (Labat & Cadéac), in dogs the size of a turkey's egg (Barrier). The wall of the aneurysm is usually thickened, although in places it may be thinned out, owing to compression by neighboring organs or from other causes. The thickening of the wall may be aided by layers of fibrin, which are sometimes deposited on its inner surface, and these may entirely obliterate the lumen of smaller vessels.

The adjoining organs are crowded aside by the constantly increasing aneurysm or they are compressed. Usually the trachea or a bronchus are found compressed, one or more vertebral bodies atrophic, the vagus or the N. recurrens compressed.

The bursting of an aneurysm almost always leads to fatal internal hemorrhage. The break may occur into the cavity of the thorax or into the abdominal cavity, or the blood may be poured into a contiguous organ to which the aneurysm had become adherent. In this manner hemorrhages may take place into the bronchi, the lungs, the stomach, the intestine or between the layers of the mesentery.

Symptoms. The symptoms of aortic aneurysm in animals have in the past not been observed with sufficient exactness. In the majority of cases the reports mention only increasing emaciation and weakness or sudden fatal internal hemorrhage.

Owing to the anatomical conditions only aneurysms which have formed on the abdominal aorta can be palpated, in larger animals from the rectum, in smaller animals through the abdominal wall. Below the vertebral column, slightly to the left, a pulsating swelling is felt, of variable size, elongated or spherical, firm or slightly elastic, which is continued into the normal segment of the aorta or into the two pelvic arteries. Sometimes the character of the swelling cannot be determined exactly; that is in cases in which edematous swellings or tough connective tissue have formed around it.

The pulsation of the swelling does not, of itself, prove the presence of an aneurysm, because tumors or abscesses connected with the aorta may show a similar pulsation, although it is then only by continuation. If it can however be determined by grasping the swelling, that it becomes harder and larger with each pulsation, an aneurysm is undoubtedly present.

An important symptom is presented by the retardation and sluggishness of the pulse, which may, however, be observed only in case of a considerable thrombosis. The pulse is felt distinctly after the heart beat, distally to the dilatation, because the copious deposit of fibrin has usually made the lumen of the vessel narrower and has thereby impeded the flow of blood toward the periphery; in consequence the increase in the systolic blood pressure, which is recognized as a pulse beat, reaches its climax, in which it can be felt, later. The vessels in which this retardation can be felt depend, of course, upon the situation of the aneurysm. If this is at the origin of the aorta, the retardation is noticed in all arteries of the body, an aneurysm in the posterior aorta causes a retardation of the pulse only in the arteries of the posterior half of the body It may also occur that the pulse is not synchronous in the two corresponding lateral halves of the body, viz., if an artery branching out from the aneurysm has become narrower.

Over aneurysms which lie superficially or which form on the aorta, vascular murmurs are usually heard, which may be perceived as either one or two murmurs or as a continued roaring. Over a pulsating swelling in the sacral region Tombari heard a blowing murmur, which had more the character of rubbing, while over a dilatation of the first segment of the posterior aorta Schmidt perceived a humming sound which was synchronous with the heart beat. In aneurysms at the origin of the aorta the murmurs are heard over the base of the heart and even lower down.

The left half of the heart is hypertrophied only in those cases in which extensive arteriosclerosis is present at the same time as the aneurysm or in which the lumen of the dilated aorta

has become much narrower *on account of thrombi.* A *dilatation* at the origin of the aorta may lead to insufficiency of the semilunar aortic valves.

In case of considerable stenosis of the vascular lumen the nutrition of the parts of the body which are distal to the dilatation is impaired. In almost all cases which have been observed for some time, weakness of the hind quarter could be determined, which was mostly noted only during motion in exceptional cases also at rest (Barrier). Finally there are symptoms of cardiac weakness (rapid fatigue, strong filling of veins, vertigo, sometimes epileptoid convulsions [Lustig]).

Nervous symptoms are observed only in exceptional cases, when a nerve trunk has been compressed by the aneurysm. In vagus compression, for instance, the heart beat may be accelerated; compression of the N. recurrens may cause laryngeal whistling. In a case observed by Schmidt the horse gave evidences of severe pain on lowering the head.

Course. A fairly long time is probably required for the development of aneurysms to that degree in which they give rise to decided morbid changes, probably several years (in Tombari's case the disease had existed at least three years). Death is due either to the compression of neighboring organs or to circulatory disturbances, unless the bursting of the aneurysm terminates life suddenly, possibly with symptoms of internal hemorrhage or with symptoms of pulmonary or intestinal hemorrhage.

Treatment. A *successful treatment of aortic aneurysm* is not possible. The stimulation of blood clots within the aneurysm by electro-puncture, injections of chloride of iron, or the introduction of foreign bodies does not enter into consideration in animals. Nor do subcutaneous injections of gelatin promise results (in man 50-130 gm., injected in one dose, of a 2-3% solution). It may be mentioned, by the way, that tetanus infection should be prevented by employing Gelatina sterilisata pro injectione (Merck). The treatment is therefore limited to saving the animals from exertion and regulating any existing cardiac disturbances. The administration of iodide of potassium also seems to be useless.

Aneurisms of other blood vessels. Even more frequently than in the aorta, a dilatation of the anterior mesenteric artery and of its main branches occurs in horses under the influence of the Sclerostomum vulgare, exceptionally and also from other causes in swine (Koch, Leibinger). In the other arteries of the abdominal cavity aneurisms are formed much more rarely, but they occur for instance in the Arteria coeliaca, the Arteria mesenterica posterior or in Arteria hepatica. In other branches of the aorta dilatations are also very rare. In Cadiot's case in an ass, and in three cases reported by Piot-Bey in cattle a dilatation of the coronary arteries had caused sudden death. Steinmeier saw a dilatation of the carotis cause sudden death by bursting and Reimers observed in the sacral region in a cow a tense, faintly pulsating bulging

which corresponded to a dilated Arteria ischiadica. Querippel described a dilatation of the crural artery, Straube one of the Arteria digitorum communis volaris, Blase one of the Arteria pharyngea in a horse 15 years old; in the last case the symptoms consisted in edema and ulcerations on the head and neck, swelling of the tongue and gums, salivation, and the usual signs of pharyngitis; the aneurism eventually burst into the pharyngeal cavity. Vogel observed a dilatation of the facial artery in a cow, Mouquet one in the cervical artery in a horse, Ravenel one in the brachial artery in a hen. Nocard found in a young dog an aneurism of the aorta and at the same time one of the pulmonary artery. Mouquet found in a horse a dilatation of the pulmonary artery; Chardin in a horse a dilatation of the hepatic artery, which weighed 14 kg. (the horse had frequently stood still on trotting and disliked to gallop), Dupas found varicose dilatations in the territory of the mesenteric veins, and Fröhner in a horse a dilatation of the external thoracic vein.

3. Obstruction of the larger arteries. Thrombosis et embolia arteriarum.

Etiology. In exceptional cases the aorta and its larger branches may be obstructed by emboli which are derived from the heart or from an aneurysm of the thoracic aorta. Usually, however, the obstruction of a relatively wider vascular lumen occurs through a thrombus originating at a spot in the wall of the vessel which has become roughened or after an embolus has become wedged in, the thrombus becoming gradually larger by deposits of new layers of fibrin. For this reason all causes which are capable of producing an endoarteritis chronica deformans may also constitute the basis of thrombosis of larger vessels. This occurs most frequently in horses and is usually brought about by the larvae of the Sclerostomum vulgare (Scl. bidentatum); in dogs the Filaria immitis sometimes leads to extensive thrombosis. Exceptionally the vascular lumen may be obstructed when arteries are compressed by a tumor (Mack, Fröhner), by exostoses (Gassner, Pirl), which may also produce chronic inflammation, even to complete occlusion of the aorta. An acute inflammation (Vennerholm) or contusion of the vessel wall (H. Bouley) may be the causes of occlusion of arteries.

Anatomical Changes. The thrombosis in horses which is usually produced by larvae of sclerostoma is found mostly in the anterior mesenteric artery, fairly frequently at the posterior end of the abdominal artery and in its pelvic branches (artt. iliacae, femorales, hypogastricae), from where the thrombus may become continued downward into the tibial arteries; in exceptional cases the vessels of an anterior extremity (art. axillaris, art. brachialis) or the first portion of the aorta (Bergstrand) is found obstructed. In a case of Filariosis haematica in a dog Hutyra found a large portion of the thoracic and abdominal aorta filled up.

The thrombus (Fig. 164) is usually attached to the roughened wall of the vessel; it is dry, tough, and usually arranged in layers. It may cover only a part of the vessel wall and thus make the lumen smaller (thrombosis parietalis), or it occupies the entire periphery of the vessel wall, in which case there remains a patent canal in the direction of its longitudinal axis

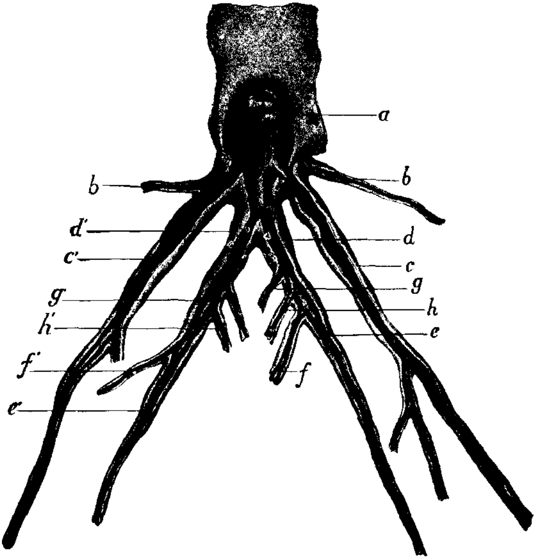

Fig. 164. **Thrombosis of the posterior end of the abdominal aorta and of its branches, in a horse.** a. Abdominal aorta; b b' Art. circumflexa ilium prof.; c c' Art. iliaca externa; d d Art. hypogastrica; e e' Art. obturatoria; f f' Art. iliolumbalis; g g' Art. glutaea sup.; h h' Art. sacralis lateralis.

(Thr. canansatus), *or finally* it obstructs the vessel entirely (Thr. obturans). The end or the outer surface of a thrombus which does not occlude the blood vessel completely is usually formed by layers of fresh fibrin, which reach into the circulating blood and perhaps into a lateral branch in the form of plugs, and are in all probability usually the result of post-mortem coagulation.

An obturating thrombus may in time disintegrate and be absorbed, but in the meanwhile a proliferation of connective

tissue from the vessel wall takes place which may eventually fill out the vessel, the lumen of which is already narrowed, completely and transform it into a firm connective tissue cord (organization of thrombus).

Pathogenesis. The obstruction of large arterial trunks produces a peculiar disturbance in the function of the muscles. In the majority of cases the vessel is not completely obstructed by the thrombus, and even if this occurs, a collateral blood circulation is established in a short time by the intermediation of branches which pass off proximally to the thrombus. The part of the body which is to be nourished by the obstructed vessel receives also in future a certain amount of blood, sufficient to supply all tissues of the body part in question with the nutritional substances necessary for life, and which sometimes even enables in muscles to accomplish a certain degree of work. If, however, the animal accomplishes prolonged and severe muscular labor, the deficiency in oxygen causes an insufficient oxidation and therefore the development of insufficient force, and also a transitory accumulation of products of metabolism in the muscle, especially lactic acid and carbonic acid, which lead to a more rapid fatigue of the muscle. When the exercise is interrupted, the products of fatigue are gradually eliminated from the muscle, the feeling of fatigue and the disturbance in function are abated, but reappear on renewed prolonged muscular exertion.

Symptoms. The form and the intensity of motor disturbances depend upon the arteries which are obstructed and upon the degree of occlusion. In the rather rare thrombosis of the posterior portion of the aorta the trotting or galloping horse begins, after a variable time, to become uncertain in the hindquarter, it staggers, the feet are raised less high, and soon it can no longer move at all, remaining standing, bathed in perspiration and trembling. If urged further, it finally collapses and falls to the ground. While lying on the ground the animal gradually becomes calmed; it arises, after a time, of its own accord and may move at a walk.

In thrombosis of aortic branches, especially the pelvic or femoral arteries, the motor disturbance commences in a similar manner, but is limited to one of the posterior extremities. After a certain time, which may be only a few minutes, the horse raises the foot less high, it cannot move it very certainly, may interfere occasionally against the other foot; it hangs its head and if possible bears the weight of the body upon the front feet; soon the joints are bent less and less, the hoof scraping the ground at each step, and finally the affected foot gives out, the animal falling upon the diseased side of the body on being urged further. In bilateral thrombosis of the femoral artery the motor disturbances correspond as a whole with those of thrombosis of the

abdominal artery, although the uncertainty in the sacral region is less striking.

Thrombosis of the axillary and brachial arteries leads to similar disturbances in motion in the one fore foot. Here also a gradually increasing muscular weakness becomes perceptible, the horse stumbling on the fore foot, knocking it against impediments and scraping the ground with the hoof, until the foot commences to tremble violently and is finally paralyzed.

Complete occlusion of the pelvic arteries causes paralysis of the sacrum, the tail, the bladder, and of the rectum; a partial thrombosis gives rise to motor disturbances, which occur only on motion and is similar to sacral paralysis.

The paralysis disappears after a certain period of rest but reappears on the next occasion, unless accidentally paralytic hemoglobinemia has been added, to which horses with thrombosis of the arteries of the extremities appear to be predisposed.

General symptoms are also present as a rule. The animal shows *great anxiety*, the heart action is remarkably excited and accelerated, the respiration is also accelerated and difficult, the mucous membranes show intense injection. In the meantime the animal breaks out in a general perspiration, but the affected extremity remains dry, and is remarkably cool to the touch. Friedberger & Fröhner have observed no general symptoms in thrombosis of the axillary artery, in spite of existing motor disturbances.

Thrombosis may be recognized with absolute certainty, if it is possible *on palpation to find firm blood clots or a compressing swelling* in the related blood vessels. In many cases the hand, which has been introduced into the rectum, may ascertain *the presence of the thrombus on palpating the aorta or the larger vessels on the sides of the pelvis*; its presence may also be assumed, owing to the weakness or complete absence of pulsation or owing to a thrill which may be felt. If the thrombosis cannot be found within the pelvis, a total or partial obstruction of the femoral or axillary arteries may be assumed to exist from the fact that the pulse cannot be felt at all or only feebly in the vessels beyond the thrombus, on the metatarsus or metacarpus.

A partial thrombosis sometimes produces no disease symptoms whatever. For instance a horse with partial thrombosis of the posterior aorta covered a distance of 130 km. two days before autopsy (Gratia).

Glöckner has observed difficult deglutition, whistling respiration, and later the symptoms of pulmonary emphysema in thrombosis of the carotid arteries.—Siegen saw epileptiform convulsions in thrombosis of the left carotid and the internal carotid; the convulsions appeared only after the horse had trotted for 20-30 minutes and lasted about 20 minutes. Cadeac observed the sudden death of a dog from thrombosis of the right carotid, following the opening of an abscess. Joest found in a dog affected with paralysis of the hind quarters a verminous aneurism in the aorta brachiocephalica, and besides a retrograding embolism of the abdominal aorta immediately anterior to its bifurcation.

Course and Prognosis. In a portion of the cases softening or contraction of attached thrombi occurs, so that the blood

paths become more or less opened, or the obstruction persists, but the circulation in the territory concerned is equalized by the collateral circulation which has meanwhile been established. In both cases the affected part of a body may after a certain time (3-6 months) again receive a sufficient supply of blood for the muscles to accomplish even continued and severe work. A complete recovery in this meaning of the term is nevertheless very rare, since the supply of blood through the narrowed paths or through the new collateral tracks still remains insufficient, and since the existing thrombosis gives rise to the further production of fibrin or to embolism, which are followed by relapses and by aggravation of the condition. In exceptional cases thrombosis of the posterior extremities may be complicated eventually by thrombosis of the brachial artery (Schraml).

In other cases, an equalization of the circulatory disturbances does not take place, the function remains permanently impaired, and the impediment to the circulation is even increased by the deposit of new masses of fibrin. In such cases the affected part of the body, usually an extremity, atrophies owing to insufficient nutrition, and the animal finally becomes unfit even for very light and slow work. In rare cases the obstruction may become so extreme that some organs receive no blood at all and die in consequence. Martin and Bouley saw, each in one case, gangrene of the muscles of the anterior extremity after obstruction of the axillary artery, Cadéac & Malet observed the same in a posterior extremity in thrombosis of the femoral artery.

If a vessel has become obstructed by an embolus derived from the first segment of the anterior or posterior aorta, the fact must be considered in giving a prognosis that similar emboli might be carried into vital organs (brain, kidneys, etc.).

Treatment. In order to promote the collateral circulation, systematic exercise of the animals is indicated, the patients being led about daily at a walk until the first symptoms of paralysis appear (Bayer). It is sometimes possible to realize even complete improvement by continued and gradually increasing work (Bayer, Johne). It may also be attempted to massage a thrombus which is palpable in the pelvic cavity, with the hand in the rectum, for 5-10 minutes daily, which proceeding may also perhaps hasten the absorption of the fibrin coagulations (Collin, Beyer). Internal treatment which has repeatedly been proposed, especially the administration of iodide of potassium, promises no results. Reichenbach observed the disappearance of lameness as early as 10 days following two intravenous injections of fibrolysin (10 gms. for each injection, at two-week intervals).

Thrombosis of the Pulmonary Artery. The obstruction of the pulmonary artery is not very rare and is brought about by emboli from the right heart. The parts of thrombi may have originated in the right heart

itself, or not infrequently they are carried there with the venous blood from distant parts of the body. In exceptional cases thrombosis forms in the pulmonary artery after the invasion of foreign bodies (hair which had been introduced on venesection from the jugular vein [Zschokke] or broken hypodermic needles [Prévot]) or in the presence of parasites (*Filaria immitis, Strongylus vasorum* [Ferez]). In many cases the cause could not be determined, probably because distant organs had not been examined carefully. Käppel succeeded in 63% of the cases diagnosed by him to find the source of the embolism by careful and systematic examination of the organs of horses which had been slaughtered (*he found thrombosis of the pulmonary artery in 9% of all slaughtered* horses, which were mostly old animals); the embolisms originated in

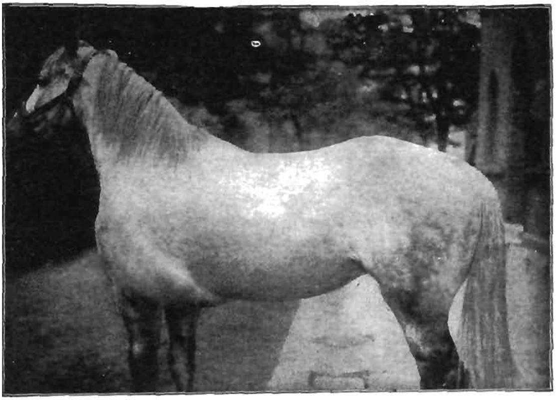

Fig. 165. **Stenosis of the anterior vena cava.** Edematous infiltration of the region of neck and shoulder; the swelling is sharply arrested near the xiphoid cartilage.

most cases in thrombotic veins in the posterior extremities and in the sheath.

Obstructions of smaller pulmonary vessels do not give rise to pathological disturbances, but when larger branches were obstructed (Käppel found this to have been the case in 12 out of 38 instances), difficult respiration occurred, like dyspnea or sudden asphyxia which *sometimes caused rapid death.*

4. Obstruction of the Large Venous Trunks. Thrombosis Venarum.

Obstruction of the large venous trunks is rare and is usually due to compression (by enlarged lymph glands, tumors,

abscesses), less often it occurs in connection with endophlebitis or slowing of the circulation.

Cases of thrombosis of the venae cavae have been observed especially in horses and cattle, once also in a dog. In the horse thrombosis of the portal vein has been known to occur.

The symptoms are easily interpreted by the anatomical arrangement of the venous trunks, because stenosis or obstruction of one vena cava produces venous stasis in that part of the body from which the vein returns the blood to the heart. Thus thrombosis of the anterior vena cava produces strong filling of the veins of head, neck, anterior extremities, and thorax, also edematous infiltration of these regions or dropsy of the chest (Fig. 165). If the obstruction is incomplete a negative pulsation may become evident in the jugular veins. Thrombosis of the posterior vena cava causes similar changes in the hindquarter, with which abdominal dropsy is associated if the location of the obstruction is between heart and liver. Thrombosis of the portal vein impairs the motility of stomach or intestine.

The circulatory disturbances may occasionally become much lessened, as was shown in a case of Albrecht's and in one of the author's.

5. Rupture of the Large Blood Vessels, Ruptura Vasorum.

The walls of the large blood vessels rupture at times, even without previous disease. The first segments of the aorta and of the pulmonary artery are very thin at the level of the semilunar valves (the aorta of the horse is in some places only 1 mm. thick, while lower down the thickness is 5-7 mm.), so that they are bulged out somewhat by the pressure of the blood, even under normal conditions (sinus valsalvae) and may rupture if a sudden rise in blood pressure occurs. Among causative factors, aside from immediate injuries, may be mentioned the pulling of heavy loads, severe exertion in galloping, vomiting, labor pains (Sigl) or other exertions. Similar causes may sometimes lead to rupture of the anterior or posterior vena cava or of the portal vein.

Usually, however, rupture is preceded by a disease of the vessel wall, although this may not be visible to the naked eye, as in a case of Caparini's. In most cases there is a sclerosis with subsequent bulging of the vessel wall, which favors rupture. This cause is responsible for the rupture of vessels, especially in older animals. The same result is observed if an abscess or ulcerating process in the immediate vicinity of the vessel is transmitted to the vessel wall from an ulcerating valve, a carious bone, or from a breaking down new growth, causing erosion of the vessel. Finally animal parasites, the larvae of Sclerostomum vulgare in the horse and Spiroptera sanguinolenta

in the dog, may weaken the vessel wall so much in places that it finally tears under the influence of the blood pressure.

The following facts may illustrate the manifold etiological factors: Palat saw rupture of the aorta at the level of the valves without demonstrable external cause, Lustig saw it occur on galloping. Sequens found in three cases the point of rupture about 1 cm. above the valves in the vicinity in which the vessel wall was diseased, but Cadiot could not find any pathological changes in two cases. In one case described by Vaerst hemorrhage occurred from a small aneurysm, situated opposite the valves.—In a case reported by Prietsch the healthy pulmonary artery was ruptured while drawing a heavy load. Block saw a four-year-old stallion bleed to death after having been thrown; the pulmonary artery was torn at the bifurcation.—Ehlers observed rupture of the portal vein after a blow on the left side of the body.—Schrulle reports a case of fatal hemorrhage from rupture of the left axillary artery in a horse which had been thrown. On autopsy of a horse Bührmann found an abscess between the pillars of the diaphragm, which had ruptured into the aorta.—In Gassner's case an exostosis on the 16th vertebra had injured the aortic wall, in Noack's case a sarcoma growing behind the kidney had done the same. Parasites may also be involved. Morgagni found in one dog, Mégnin in three dogs, the Spiroptera sanguinolenta in the wall of the aorta; it was present in the form of small whitish nodes one of which had caused rupture of the aorta. Durieux and Degive finally could demonstrate in a horse that fatal hemorrhage had been due to destruction of tissue by the larvae of the Sclerostomum vulgare (in one case the aorta was apparently sound, in the other it presented in its entire length the picture of atheromatosis).

The symptoms of rupture of a vessel indicate in general only internal hemorrhage, the exact cause of which can usually only be ascertained on autopsy.

Index, Vol. II

	PAGE
Abortion, infectious, Vol. I......	
Abcess in the esophagus........	49
Abcess in the liver.............	446
Abcess in the lung............	699
Acanthocephala	356
Acarus, farinæ..................	2
Acetonemia in cattle...........	105
Actinobacillosis, Vol. I..........	
Actinomycosis, Vol. I...........	
Actinomycosis of the esophagus.	68
Actinomycosis of the intestines..	250
Actinomycosis of the larynx....	581
Actinomycosis of the liver......	463
Actinomycosis of the nasal membranes	545
Actinomycosis of the peritoneum	518
Actinomycosis of the stomach...	159
Actinomycosis of the throat.....	45
Actio cordis intermittens.......	760
Adams-Stokes Syndrome........	763
Adenocarcinoma of the intestines	250
Adenocarcinoma of the liver.....	461
Adenocarcinoma of the lungs....	713
Adenocarcinoma of the stomach.	159
Adenoma of the heart..........	794
Adenoma of the intestines......	250
Adenoma of the liver...........	462
Adenoma of the lungs...........	713
Adenoma of the stomach........	159
Aegagropili	109
Agglutination, Vol. I............	
Aggressins, Vol. I.............	
Albuminuria, Vol. III...........	
Allantiasis	212
Allergy, Vol. I.................	
Allorrhythmia	657
Ameba meleagridis	420
Amebic dysentery	422
Amphistoma Collinsi............	355
Amphistomum conicum	331
Amphistomum explanatum......	491
Amphistomum truncatum	491
Amyloid degeneration...........	434
Amyloid induration.............	434
Amyloid liver...................	434
Amyloid reaction...............	435
Amyloidosis hepatis.............	434
Anaplasmosis, Vol. III...........	
Anchylostomiasis	374
Anchylostomum bovis...........	394
Anchylostomum duodenale......	382
Anchylostomum radiatum.......	382
Anemia, Vol. III...............	
Aneurism of the aorta.........	824
Aneurism of the blood vessels...	827
Aneurism of the coronary arteries	773
Aneurism of the heart, acute....	798
Aneurism of the heart, chronic..	798
Aneurism of the heart, fibrous..	790
Aneurism, rupture	798
Aneurism, valvular	798
Aneurism, Vol. I...............	
Angina 30, 31,	569
Angina diphtheritica, Vol. I.....	
Angina membranacea..........	575
Angioma of the heart...........	794
Angioma of the liver............	462
Angioma of the nasal cavities...	545
Angioma of the peritoneum.....	518
Anguillula stercoralis...........	398
Anoplocephala mamillana.......	344
Anoplocephala perfoliata........	344
Anoplocephala plicata..........	344
Anthrax, Vol. I...............	
Aorta, aneurism of.............	824
Aorta, calcification of..........	820
Aorta, rupture of...............	834
Aphonia	581
Aphtha, epizootic, Vol. I.........	
Aphtha, sporadi................	9
Arhytmis cordis................	760
Arteriosclerosis	820
Arteritis, acute.................	828
Arteritis, chronic...............	820
Artery, compression of.........	828
Artery, crushing of.............	828

PAGE

Artery, plugging of 828
Artery, celiac, occlusion of 271
Arthritis urica, Vol. III........
Artificial feeding 43
Arythmatic action of the heart, systolic 762
Arythmatic action of the heart, true 762
Ascariasis 356
Ascariadæ in the intestines..... 356
Ascariadæ in the liver.......... 496
Ascaris lumbricoides 357
Ascaris marginata.......... 357
Ascaris megalocephala......357, 496
Ascaris mystax 358
Ascaris ovis.......... 357
Ascaris suilla.......... 357
Ascaris vituli.......... 357
Ascites adiposus.......... 502
Ascites chyliformis.......... 502
Ascites chylosus.......... 502
Aspergillus 695
Aspergillus in birds.......... 510
Aspergillus fumigatus.......... 510
Aspergillus glaucus.......... 695
Aspergillus niger.......... 695
Aspergillus nigrescens.......... 695
Asthma, bronchial.......... 594
Asthma, dyspeptic.......... 594
Asthma, nasal.......... 525
Asthma, nervous.......... 594
Asthma, spasmodic.......... 594
Atelectasis 586, 605, 682
Atony of rumen, reticulum and amasum 95
Atony of rumen in young calves. 101
Atrophy of heart.......... 790
Atrophy, yellow of liver........ 441
Atrophy, pancreatic.......... 499
Atrophy, senile.......... 632
Atropin 637

Bacillus bipolaris septicus..646, 685
Bacillus botulinus.......... 213
Bacillus bovis morbificans...... 213
Bacillus bronchiolitidis vituli.... 685
Bacillus coli communis.......... 200
Bacillus dysenteriæ.......... 211
Bacillus enteritidis.......... 200
Bacillus fusiformis.......... 26
Bacillus necrophorus.......... 32
Bacillus nodulifaciens bovis, Vol. I..........
Bacillus pneumoniæ felis........ 647
Bacillus pneumoniæ tigris...... 647
Bacillus pyocyaneus.......... 533
Bacillus pyogenes..........10, 24
Bacillus salivarius septicus...... 21
Bacillus septicæmiæ anserum, Vol. I..........

PAGE

Bacterium subtile agnorum...... 25
Balantidium coli.......... 423
Bezoar 109
Bile coloring matter in the urine 426
Black-head 419
Black-water fever, Vol. I........
Blenorrhea, nasal.......... 536
Bloating, acute, of ruminants... 86
Bloating, chronic, of ruminants.. 93
Blood, diseases of, Vol. III......
Blood parasites, Vol. III........
Blood vessel, rupture of......... 834
Blood vessels, diseases of...... 820
Bombyx processionalis.......... 2
Bothriocephalus cordatus....... 349
Bothriocephalus felis.......... 352
Bothriocephalus fuscus.......... 349
Bothriocephalus latus.......... 349
Botryomycosis of the lungs..... 713
Bots, of stomach.......... 314
Bottom disease.......... 450
Botulism 212
Botulism toxin.......... 213
Bradycardia 759
Bronchial asthma.......... 594
Bronchial catarrh.......... 583
Bronchial catarrh of birds...... 588
Bronchial croup.......... 592
Bronchial diseases.......... 583
Bronchiectasis 586
Bronchiolitis 586
Bronchitis and broncho-pneumonia in horses, infectious 678
Bronchitis, capillary.......... 586
Bronchitis, catarrhal.......... 583
Bronchitis, croupous.......... 592
Bronchitis, dry.......... 587
Bronchitis, putrid..........586, 589
Bronchitis, sclerotic.......... 589
Bronchitis, verminous.......... 595
Broncho-pneumonia 671
Broncho-pneumonia, enzootic of young animals.......... 684
Broncho-pneumonia, fibroplastic. 708
Broncho-pneumonia, infectious, of lambs.......... 684
Broncho-pneumonia, pseudotubercular 605
Broncho-pneumonia, purulent, of geese 672
Broncho-pneumonia, verminous.. 595
Broncho-pneumonia, verminous diffuse 604
Broncho-pneumonia, verminous lobar 604
Broncho-pneumonia, verminous lobular 604
Broncho-stenosis 593
Brussels disease 678

PAGE
Cadaverin 199
Cancer of the liver 461
Carcinoma hepatis 461
Carcinoma of the intestines 250
Carcinoma of the larynx 581
Carcinoma of the liver 461
Carcinoma of the lungs 713
Carcinoma of the nasal cavities 545
Carcinoma of the peritoneum 519
Carcinoma of the stomach 159
Carcinoma of the throat 581
Catarrhal fever malig., Vol. I...
Catarrhus antri Highmori 556
Catarrhus bronchialis 583
Catarrhus intestinalis acutus 170
Catarrhus intestinalis chronicus. 190
Catarrhus laryngis 569
Catarrhus narium 523
Catarrhus sacci aërophori 564
Catarrhus sinus frontalis 562
Catarrhus ventriculi acutus 124
Catarrhus ventriculi chronicus 132
Cella infraorbitalis, inflammation of 560
Cement cough 684
Cephalomyia ovis 584
Cephalomyia rufibarbis 551
Cephalomyia stimulator 551
Cephalomyia trompe 551
Cephalomyia ulrichii 551
Cephenomya trompe 44
Cercaria 471
Cercomonas hepaticum 496
Cestodes, see Tænia 342
Cetonia aurata 401
Cheiracanthus hispidus 332
Chemotherapy, Vol. I..........
Chlamydozoa, Vol. I..........
Choleithiasis 420
Cholera of fowls, Vol. I..........
Cholera of swine, Vol. I..........
Cholin 199
Cirrhosis hepatis 450
Cirrhosis hepatis atrophica 450
Cirrhosis hepatis hyperplastica 450
Cirrhosis pulmonum 707
Coccidiosis hepatis 494
Coccidiosis intestinalis 403
Coccidiosis intestinalis avium 416
Coccidiosis intestinalis bovum 403
Coccidiosis intestinalis canum 413
Coccidiosis intestinalis cuniculi 413
Coccidiosis intestinalis caprarum 410
Coccidiosis intestinalis ovum 410
Coccidium Arloingi 410
Coccidium avium 416
Coccidium bigeminum 413
Coccidium Faurei 410
Coccidium oviforme 413, 494
Coccidium perforans 414
Coccidium Pfeifferi 416
Coccidium tenellum 416
Coccidium truncatum 416
Coccidium Zurni 404
Colic 307
Colic, catarrhal 187
Colic, distension 213
Colic, false 309
Colic, flatulent 213
Colic, habitual 252, 269
Colic, impaction 219
Colic, periodic 252, 264
Colic, rheumatic 187
Colic, symptomatic 265
Colic, thrombo-embolic 256
Colic, wind 255
Combined valvular lesions 813
Complement fixation in echinococcosis 468
Compression of the heart 754
Compressio oesophagi 59
Concrementa sacci aërophori 565
Congestion, intestinal 167, 202
Coprolith 138
Coprostasis 219, 232
Cor adiposum 793
Cor villosum 741
Corpora alieni reticuli, ruminis et omasi 108
Corpora alieni ventriculi 152
Coryza 526
Coryza of birds 529
Coryza contagious 529
Coryza gangrenous, Vol. I..........
Coryza ozena 526
Coryza, pustular, of the horse 541
Cough, enzootic 573
Crop, animal parasites of 73
Crop, hard 70
Crop, incision of 71
Crop, soft 70
Crotalariosis equorum 640
Croup 575
Croup of Cattle, Vol. I..........
Cryptocystis trichodectis 348
Cyathostumum 386
Cyathostumum tetracanthum 386
Cysticercosis hepatis 491
Cysticercus 491
Cysticercus fasciolaris 352
Cysticercus pisiformis 347, 491
Cysticercus tenicollis 491, 713
Cysticercus tenuicollis in the abdominal cavity 519
Cysticercus tenuicollis in the liver 491
Cysticercus tenuicollis in the lungs 713
Cytodites nudus 616
Cytoleichus sarcoptoides 616

PAGE
Davainea carioca.............. 353
Davainea cataniana............ 353
Davainea cesticillus............ 353
Davainea crassula............. 353
Davainea echinobothrida........ 353
Davainea proglottina.......... 353
Decomposition in heart valve defects 798
Degeneratio adiposa hepatis..... 432
Degeneratio amyloidea hepatis.. 434
Dengue fever, Vol. I...........
Dermoid cysts in the lungs..... 713
Diabetes insipidus, Vol. I......
Diabetes mellitus, Vol. I........
Diafarin 135
Diagnosis of parasitic diseases of the stomach and intestines... 339
Diaphragm, hernia of...253, 273, 279
Diaphragm, rupture of.......... 88
Diarrhea, chronic.............. 190
Diastasolin 135
Dicraniotænia echinobothrida... 353
Dicraniotænia sphenoides....... 353
Dicrocœlium lanceolatum....... 469
Dilation of the Aorta.......... 829
Dilatatio cordis................ 777
Dilatatio cordis activa.......... 778
Dilatatio cordis passiva......... 778
Dilatatio oesophagi............ 63
Dilatatio ruminis ab alimentis... 80
Dilatatio ventriculi acuta...136, 147
Dilatatio ventriculi chronica..... 149
Diphtheria, Vol. I.............
Diplococcus pneumoniæ......... 648
Diplococcus capsulatus.......... 199
Diplospora 415
Dipylidium caninum............ 348
Dipylidium Chyzeri 352
Dispharagus laticeps........... 352
Dispharagus nasutus............ 335
Dispharagus uncinatus 73, 337
Dissociation of the heart....... 763
Distoma commutatum........... 356
Distoma eggs in feces.......... 340
Distoma ovatum 356
Distomatosis 468
Distomatosis of the lungs....... 712
Distomum albidum............. 491
Distomum campanalatum....... 491
Distomum coniunctum.......... 491
Distomum felinum.............. 491
Distomum hepaticum.......... 469
Distomum hepaticum in the abdominal cavity.............. 520
Distomum hepaticum in the heart 795
Distomum lanceolatum......... 469
Distomum pulmonale........... 712
Distomum truncatum........... 491
Disturbances in the circulation, acute 776

PAGE
Dithyridium 520
Dithyridium elongatum......... 520
Diverticulum 64
Diverticulum of the intestine... 251
Diverticulum of the oesophagus. 64
Diverticulum of the oesophagus, false 64
Diverticulum of the oesophagus, true 64
Dochmiasis 374
Dochmiasis in carnivora........ 374
Dochmiasis in cattle............ 381
Dochmiasis in silver foxes...... 380
Dochmiasis in cats and feline beasts of prey................ 380
Dochmiasis in sheep and other animals 385
Dochmiasis, larval............. 393
Dochmius cernuus............. 385
Dochmius duodenalis........... 374
Dochmius longemucronatus..... 385
Dochmius radiatus............. 382
Dochmius stenocephalus........ 375
Dochmius trigonocephalus...... 374
Dourine, Vol. I................
Drepanidiotænia anatina........ 353
Drepanidiotænia cantaniana..... 353
Drepanidiotænia coronula....... 353
Drepanidiotænia fasciata........ 353
Drepanidiotænia gracilis........ 353
Drepanidiotænia infundibuliformis 353
Drepanidiotænia lanceolata...... 353
Drepanidiotænia setigera........ 353
Drepanidiotænia sinuosa........ 353
Dysentery 211, 212
Dysentery of adult cattle....... 197
Dysentery, coccidial of cattle.... 403
Dysentery, red, of cattle........ 403
Dysentery, white, of fowls..... 416
Dyspepsia, acute............... 124
Dyspepsia, chronic.............. 95
Dysperistaltica 288

Ear drum perforation........... 567
Ecchondroma in the nasal cavities 545
Echinococcus in the heart...... 795
Echinococci in the liver......... 463
Echinococci in the lungs........ 710
Echinococci in the peritoneal cavity 520
Echinococcosis hepatis.......... 463
Echinococcosis pulmonum....... 710
Echinococcus alveolaris.....347, 467
Echinococcus cysticus.......... 467
Echinococcus endogenus........ 465
Echinococcus exogenus......... 465
Echinococcus granulosus........ 465
Echinococcus hydatitosus....... 465

	PAGE
Echinococcus multilocularis	464
Echinococcus polymorphus	347
Echinococcus secundarius	465
Echinorhynchus filicollis	401
Echinorhinchus canis	403
Echinorhynchus gigas	401
Echinorhynchus polymorphus	403
Echinorhynchus sphærocephalus	404
Echinostomum perfoliatum	355
Ectasia oesophagi	63
Edema of the larynx	579
Eimeria cuniculi	413
Eimeria Zürni	404
Emesis	74
Emphysema alveolar	627
Emphysema pulmonary, alveolar	627
Emphysema pulmonary, alveolar acute	627
Emphysema pulmonary, alveolar genuine	630
Emphysema pulmonary *intersti-tial*	639
Emphysema pulmonary vicarians	627
Empyema	718
Empyema antri Highmori	562
Empyema sacci aërophori	564
Empyema sinus frontalis	562
Empyema sinus maxillaris	556
Endoarteritis chronica	820
Endocarditis acuta	795
Endocarditis, chronica	803
Endocarditis, parietal	798
Endocarditis, ulcerous	798
Endocarditis, valvular	798
Endocarditis, verrucous	798
Enchondrosis of the cricoid carti-lage	581
Enteralgia	187
Enteralgia catarrhal	187
Enteritis in birds	210
Enteritis, caseous, Vol. I	
Enteritis, catarrhal	170
Enteritis, croupous 194,	197
Enteritis, croupous, of cats	197
Enteritis, croupous, of horses	194
Enteritis, membranous......172,	194
Enteritis, mycotic	197
Enteritis, septic	197
Enteritis, typhoid	197
Enterodynia	307
Entero-hepatitis, infectious	419
Enteroliths	239
Enterorrhagia	167
Enzootic tumors in the region of the ethmoid bone	546
Epistaxis	521
Epizootic tracheal catarrh	573
Erosions, hemorrhagic	214
Eructation	77
Esophagitis	48
Eustrongylus gigas, Vol I	
Eustrongylus gigas in the liver	463
Fasciola hepatica	469
Fasciola lanceolata	469
Fatty degeneration	432
Fatty heart	793
Fatty infiltration	432
Fatty liver	432
Feeding, artificial	43
Fibroma of the intestines	250
Fibroma of the lungs	713
Fibroma of the nasal cavity	545
Fibroma of the peritoneum	519
Fibroma of the stomach	159
Filaria labio papillosa	519
Filaria nasuta	335
Filaria papillosa 401,	519
Filaria tracheobronchialis	599
Filaria uncinata	337
Flexion of the cecum	289
Flexion of the colon	288
Fluke, liver	468
Follicular inflammation, nasal	541
Food balls in the rumen	108
Food balls in the stomach	152
Foramen interventriculare per-sistens	804
Foreign bodies in the bronchi	593
Foreign bodies in the esophagus	53
Foreign bodies in the gall ducts	431
Foreign bodies in the intestines	239
Foreign bodies in the liver	431
Foreign bodies in the pericar-*dium* 739,	742
Foreign bodies in the rumen	108
Foreign bodies in the stomach	152
Foreign bodies in the stomachs of carnivora	152
Foreign bodies in the stomachs of ruminants	108
Fungus poisoning	197
Fungus poisoning, differential di-agnosis	207
Gadflies	547
Gallstone colic	430
Gangrene, pulmonary	701
Gapes	614
Gas formation in the thorax	661
Gas poisoning in battle	622
Gas in pericardium	756
Gastritis, catarrhal	132
Gastritis hyperplastica of horses	136
Gastritis, phlegmonous	204
Gastritis, polypous	133
Gastritis, proliferative	133
Gastritis, purulent	204
Gastritis, traumatic	222
Gastritis, verrucous	133

Gastrodiscus Aegyptiacus....... 355
Gastro-enteritis 183, 197
Gastro-enteritis dysenteric...... 197
Gastro-enteritis hæmorrh. bovum 204
Gastro-enteritis hæmorrh. canum 204
Gastro-enteritis mycotic......... 197
Gastrolith 152
Gastromycosis ovis, Vol. I......
Gastrophilus 314
Gastrophilus equi............44, 314
Gastrophilus flavipes............ 315
Gastrophilus hemorrhoidalis..44, 314
Gastrophilus inermis............ 315
Gastrophilus nasalis............ 315
Gastrophilus pecorum........... 48
Gigantorhinchus hirudinaceus... 401
Gingivitis 202
Glottis edema.................. 579
Gnathostoma hispidum......... 332
Going light..................... 190
Gongylonema pulchrum......... 70
Gongylonema scutatum......... 70
Gregarinæ rhinitis.............. 555
Grippe 573
Guttural pouches, catarrh of.... 564
Gutturoliths 565

Hæmopericardium 755
Hair balls in the intestines..... 239
Hair balls in the rumen........ 108
Hair worm disease............ 600
Hæmangioma cavernosum....... 462
Hæmopis sanguisuga........... 45
Hæmoptœ 624
Hay fever..................... 525
Heart atrophy 790
Heart defects.................. 757
Heart defects, congenital....... 813
Heart dilatation 777
Heart fatty..................... 793
Heart hypertrophy............. 783
Heart insufficiency............ 804
Heart muscle, calcification of.... 792
Heart muscle, inflammation of, acute 785
Heart muscle, inflammation of chronic 789
Heart muscle, inflammation of parenchym. 786
Heart muscle, inflammation of purulent 786
Heart valvular defects, compensating 813
Heart valvular defects, noncompensating 815
Heart weakness 764
Heart weakness, acute......... 765
Heart weakness, chronic........ 765
Heaves 630
Helix carthusiana............. 471
Helminthiasis 338

PAGE
Helminthiasis, larval........... 393
Helminthiasis, nodular.......... 393
Hematemesis 165
Hematozoa, Vol. I............
Hematuria, Vol. III...........
Hemiclepsis tespellata.......... 554
Hemistoma alatum............. 355
Hemoglobinemia, Vol. III........
Hemoglobinuria, Vol. III........
Hemophilia, Vol. III...........
Hemorrhagia hepatis 436
Hemorrhagia narium........... 521
Hemorrhagia ventriculi......... 165
Hemorrhoids 167
Hepar adiposum 432
Hepar Amyloidium............. 434
Hepatic coccidiosis............. 494
Hepatis acuta traumatica distomatosa 468
Hepatitis cysticercosa.......... 491
Hepatitis enzootica porcellorum. 441
Hepatitis fibrosa............... 450
Hepatitis hemorrhagica mortificans 441
Hepatitis indurativa............ 450
Hepatitis indurativa distomatosa 478
Hepatitis interstitialis diffusa chronica 450
Hepatitis parenchymatosa acuta 438
Hepatitis parenchymatosa acuta diffusa ovum 439
Hepatitis suppurativa 446
Hernia, external............... 253
Hernia, internal............... 251
Heterakiasis 368
Heterakis brasiliensis.......... 368
Heterakis compressa........... 368
Heterakis differens............ 368
Heterakis dispar............... 368
Heterakis inflexa.............. 368
Heterakis maculosa............ 368
Heterakis papillosa............ 368
Heterakis perspicillum.......... 368
Heterakis vesicularis 368
Holostomum gracile............ 355
Hordeum silvaticum............ 28
Hornworm disease............. 547
Horse typhoid................. 678
Hydatids in the liver........... 463
Hydremia of cattle, Vol. III.....
Hydronephrosis, Vol. III........
Hydropericardium 754
Hydropneumothorax 735
Hydrops antri Highmori........ 556
Hydrops ascites............... 501
Hydrops ascites adiposus....... 502
Hydrops ascites chyliformis..... 502
Hydrops ascites chylosus....... 502
Hydrops ascites parasitarius ... 520
Hydrops pericardii.......... .. 754

PAGE

Hydrops sacci aërophori........ 565
Hydrops sinus maxillaris....... 556
Hydrothorax 732
Hyperemia, pulmonary.......... 618
Hyperplasia mucosæ narium.... 545
Hyperplasia polyposa mucosæ laryngis 581
Hypertrophy of heart muscle... 783
Hypertrophy, idiopathic........ 783
Hypertrophy, nephritic......... 784
Hypertrophy, pathologic........ 783
Hypertrophy, physiologic........ 783
Hypertrophy, secondary......... 784
Hyphomycosis destruens........ 19
Hypoderma in the esophagus... 49
Hyptiasmus tumidus........... 555

Ichthyosism 212
Icterus 424
Ictrogen 443
Ileitis catarrhalis acuta........ 175
Ileus 77
Immune serum, polyvalent 694
Immunization, Vol. I..........
Impaction 219
Incarceration of the intestine... 272
Indican in the urine in obstipation 225
Indican in the urine in dilatation of the stomach.............. 141
Indigestion, acute.............. 80
Indigestion, chronic............ 95
Indigestion, chronic, of ruminants 95
Indigestion, gaseous........... 86
Indigestion, ingluvial.......... 70
Indigestion, intestinal.......... 213
Indigestion, mephitic........... 86
Indigestion, simple............ 99
Indigestion in the stomach..... 136
Indigestion, traumatic.......... 108
Induratio brunea pulmonum..... 620
Infarct, hemorrhagic in the intestines 261
Infectious Bronchitis and Bronchopneumonia in horses...... 678
Inflammation, ingluvial......... 70
Influenza, Vol. I..............
Influenza bacillus.............. 536
Influenza of cattle............. 573
Influenza-like disease of cattle... 573
Influenza-like disease of rabbits.. 536
Infra-orbital cavity, inflammation of 562
Infusions in the rectum........ 187
Infusions in the veins.......... 43
Ingluvitis 70
Inhalation 528, 572
Injections, intranasal 42
Injections, intrapharyngeal 42
Injections, intratracheal 572, 612

PAGE

Insufficiency, muscular 804
Insufficiency, relative 804
Insufficiency, valvular bicuspid.. 808
Insufficiency, valvular tricuspid . 811
Insufficiency, valvular, semilun aortae 810
Insufficiency, valvular, semilun art. pulm. 805
Intermission of heart beats...... 760
Intestinal calculus 240
Intestinal catarrh, acute 170
Intestinal catarrh, chronic 190
Intestinal catarrh, infectious, in birds 172
Intestinal catarrh, infectious, in swine 172
Intestinal coccidiosis in birds.. 416
Intestinal coccidiosis in carnivora 413
Intestinal coccidiosis in cattle.. 403
Intestinal coccidiosis in rabbits. 413
Intestinal coccidiosis in sheep and goats 410
Intestinal compression 250
Intestinal constriction 249
Intestinal croup 194, 197
Intestinal diseases 167
Intestinal hemorrhage 167
Intestinal incarceration 272
Intestinal injuries 250
Intestinal intussusception 299
Intestinal invagination 299
Intestinal meconium 241
Intestinal meteorism 213
Intestinal mycosis 197
Intestinal occlusion, internal ... 219
Intestinal pain, catarrhal 187
Intestinal resection 271
Intestinal spasm, catarrhal 187
Intestinal stenosis 249
Intestinal strangulation 272
Intestinal stricture 249
Intestinal torsion 286
Intestinal typhoid 197
Intestinal volvulus 286
Intestinal worms 338
Intussusceptio intestini 299
Invaginatio intestini........... 299
Invaginatio intestini agonalis ... 301
Irrigation of rectum 181
Irrigation of rumen 105

Jagziekte of horses 640
Japp disease of lambs.......... 575
Jaundice 424
Jejunitis catarrhalis acuta..... 175

Kidnēy diseases, Vol. III ......
Kolumbácser flies 44
Kum Gata disease 230

	PAGE
Lachnosterna	401
Laminosioptes cysticola	617
Laparo-enterotomy	248
Laparo-gastrotomy	
Laparotomy test	285
Laparotomy 286, 307,	313
Laryngeal catarrh, epizootic.573,	574
Laryngeal catarrh, infectious	569
Laryngeal compression	582
Laryngeal diseases	569
Laryngeal edema	579
Laryngeal tumors	587
Laryngitis, catarrhal	569
Laryngitis, croupous	575
Laryngitis, fibrinous	575
Laryngitis, granular	570
Leiomyoma of the intestines	250
Leptothrix buccalis	715
Leukemia, Vol. III	
Leukemia of fowls, Vol. III	
Limnæus minutus	472
Limnæus pereger	472
Limnæus truncatulus	472
Lingulatula denticulata 495,	712
Lingulatula in the liver	495
Lingulatula tænioides	552
Linguatulosis nodularis	397
Lipoma in the intestines	250
Lipoma in the larynx	581
Lipoma in the liver	463
Lipoma in the lungs	713
Lipoma in the nasal cavities	545
Lipoma in the pharyngeal cavity	45
Lipoma in the stomach	159
Lipoma of the heart	794
Lipoma on the peritoneum	519
Liver, abscess of	446
Liver, actinomycosis of	463
Liver, amyloid	434
Liver, atrophy, acute yellow	441
Liver, cancer of	461
Liver, cirrhosis of	450
Liver, coccidiosis of	494
Liver, cysticercosis of	491
Liver, diseases of	424
Liver, echinococcosis of	463
Liver, excision of	462
Liver, fatty degeneration of	432
Liver, fluke disease	468
Liver, foreign bodies in	431
Liver, incision	448
Liver, induration	450
Liver, necrosis of	448
Liver, parasites of	495
Liver, rupture of	436
Liver, sclerostomiasis of	495
Liver, tuberculosis	463
Liver, tumors of	462
Lombriz, Vol. I, Vol. III	
Lumbago, Vol. III	
Lung, abscess of	699
Lung, atelectasis of	682
Lung atrophy, senile	631
Lung, cysticercosis of	713
Lung distomatosis	712
Lung echinococcosis	710
Lung emphysema, acute	627
Lung emphysema, alveolar	627
Lung emphysema, chronic	630
Lung emphysema, interstitial	639
Lung emphysema, simple	627
Lung, gangrene of 660,	701
Lung, hair worms in	600
Lung, hemorrhage from	624
Lung inflammation, see pneumonia	643
Lung, parasites of	712
Lung, pentastomes in	712
Lung, phthisis verminous	595
Lung, tumors of	713
Lung worm diseases	595
Lupine disease	443
Lupinosis	443
Lupinus alba	443
Lupinus cœruleus	443
Lupinus flavus	443
Lymphadenitis of abomasum	159
Lymphadenitis of rumen	159
Lymphangioma of heart	794
Lymphangioitis epizootica, Vol. 1	
Lymphangioitis farciminosa bovis, Vol. I	
Lymphangioitis pseudofarcinosa, Vol. I	
Lymphosarcoma in the larynx	581
Lysis	656
Lyssa, Vol I	
Macro-bronchitis	586
Mal de Caderas, Vol. I	
Malignant strangles	678
Malta fever, Vol. I	
Massage of intestine through rectum	234
Massage of rumen for removal of foreign bodies	122
Maxillary sinus, catarrh of	556
Meat poisoning 197,	212
Meconotorium	247
Melanoma of the esophagus	68
Melanoma of the intestines	250
Melanoma of the larynx	581
Melanoma of the liver	462
Melano-sarcoma of heart	794
Melano-sarcoma of the intestines	250
Melano-sarcoma of the liver	462
Melano-sarcoma of the lungs	713
Mercurial stomatitis	23
Mesenteric artery, occlusion of	256

PAGE
Mesenteric vein, occlusion of.... 272
Mesocestoides lineatus 349
Meteorism, acute 86
Meteorism, chronic, in calves... 101
Meteorism, chronic, in ruminants 95
Meteorism, circumscribed 266
Meteorism, intestinal 213
Meteorism, local 262-276
Methylguanidin 199
Micro-bronchitis 586
Micrococcus ambratus 684
Molluscum contagiosum, Vol. I.
Moniezia alba 345
Moniezia Benedeni 345
Moniezia denticulata 345
Moniezia expansa 345
Moniezia trogonophora 346
Monilia candida 17
Monostomum attenuatum 355
Monostomum caryophyllinum ... 355
Monostomum flavum 617
Monostomum verrucosum 355
Mosaic liver 492
Mouth, catarrh of 1
Mouth, diseases of 1
Mouth, inflammation of 6
Mucor conoideus 696
Mucor mucedo 696
Mucor racemosus 696
Mucor spinosus 557
Muguet 17
Mumps 28
Muscarin 199
Mycosis intestinalis 197
Mycosis, primary 695
Mycosis, secondary 695
Mycosis, true 695
Myocarditis, acute 785
Myocarditis, chronic 789
Myocarditis, embolic 786
Myocarditis, parenchymatous ... 787
Myocarditis, purulent 786, 787
Myocarditis, septic 786
Myoma of the intestines 250
Myoma of the stomach 159
Myxoma in the heart........... 794
Myxoma in the intestines....... 250
Myxoma in the nasal cavities... 545
Myxoma on the peritoneum...... 519

Nasal catarrh 523
Nasal catarrh, benign, of cattle.. 529
Nasal catarrh, contagious, of cats 539
Nasal catarrh, infectious, of fowls 529
Nasal catarrh, infectious, of geese 536
Nasal catarrh, malignant, of swine 532
Nasal cavity, parasites of 547

PAGE
Nasal coccidiosis 555
Nasal croup 540
Nasal diseases 521
Nasal flies 314
Nasal hemorrhage 521
Nasal inflammation 523
Nasal irrigation 528
Nasal membrane, necrosis of... 541
Nasal tumors 545
Nasal vein, thrombosis of...... 523
Necrosis nodosa hepatis......... 448
Nemathelminths 356
Nematodes 356
Nematodes in stomach of horse.. 322
Nematodes in lungs of sheep... 595
Neoplasms, see tumors.
Nephritis, Vol III..............
Neuralgia colica 307
Neurin 197
Noma 25
Non compensating heart valves.. 815
Nourishment, artificial 43

Obesity, Vol. III ....
Obstipation 219, 232
Obstruction of the large arteries 828
Obstruction, esophageal 53
Obstruction, ingluveal 70
Obturatio intestini 239
Occlusio oesophagi 53
Oesophagostomum 300, 393
Oesophagostomum radiatum 394
Oesophagostomum venulosum .. 394
Oesophagostomum dentatum 394
Oesophagus, See Esophagus.
Œstrus maculatus 551
Œstrus ovis 547
Œstrus purpureus 44, 551
Œstrus rhinoestrus 551
Oidium albicans 17
Ollulanus tricuspis 599, 335
Omastitis 95, 123
Omentopexia 508
Opsonine, Vol. I ....
Ossification of the heart muscle 792
Osteomata 545, 713
Oulitis 3
Oxyuriasis 371
Oxyuris ambigua 371
Oxyuris curvula 371
Oxyuris equi 371
Oxyuris mastigodes 371
Oxyuris vermicularis 371
Ozena 526

Palisade worms 386
Palisade worms in horses....... 386
Palisade worms in the intestines 386
Palpatio cordis 757
Pancreas, diseases of 497

PAGE
Papilloma of the esophagus..... 68
Papilloma of the pharnyx 45
Paracholia 425
Paragonimus Westermanni 712
Paralysis of the esophageal nerves 52
Paralysis of the pharyngeal nerves 39
Parapedesis 425
Parasites, animal, of the abdominal cavity 519
Parasites, animal of the air passages in birds 614
Parasites, animal, of birds...... 335
Parasites, animal, of the blood, Vol. I ....
Parasites, animal, of the bronchi 595
Parasites, animal in the heart.. 795
Parasites, animal, of carnivora . 334
Parasites, animal, of cattle 330
Parasites, animal, of the crop.. 73
Parasites, animal, of the esophagus 70
Parasites, animal, of goats 323
Parasites, animal, of horses.... 314
Parasites, animal, of the intestines 338
Parasites, animal, of the liver.. 463
Parasites, animal, of the lungs. 595
Parasites, animal, of the nasal cavities 547
Parasites, animal, of the pharynx 44
Parasites, animal, of sheep..... 323
Parasites, animal, of the stomach 314
Parasites, animal, of swine 332
Parotitis 28
Pellicula linguæ 4
Penicilia 695
Pentastomum denticulatum 495, 712
Pentastomum of the peritoneal cavity 519
Pentastomum of the liver....... 495
Pentastomum of the lungs 712
Pentastomum tænioides 551
Periarteritis nodosa 824
Pericarditis circumscripta 741
Paricarditis diffusa 741
Pericarditis fibrosa adhesiva ... 742
Pericarditis traumatica 739, 742
Peripneumonia 701
Peribronchitis nodosa 586, 600
Pericarditis 738
Pericarditis externa 509
Peritonitis 509
Peritonitis acuta 513
Peritonitis chemica 510
Peritonitis chronica 511
PAGE
Peritonitis, chronica circumscripta 515
Peritonitis diffusa acuta 513
Peritonitis fibrinosa 513
Peritonitis hemorrhagica 511
Peritonitis operativa 509
Peritonitis perforativa 509
Peritonitis purulenta 514
Peritonitis rheumatica 511
Peritonitis traumatica 509
Perityphlo-hepatitis meleagridum 420
Pestis avium, Vol. I............
Pestis bovum orientalis, Vol I.
Pestis equorum, Vol. I.........
Pestis suum, Vol. I.............
Pharyngitis 31
Pharyngitis, catarrhal 34
Pharyngitis, croupous 35
Pharyngitis, diphtheritic 35
Pharyngitis, purulent 34
Physocephalus sexalatus 334
Phytoconcrementa 220
Pictou cattle disease 451
Pituitas 4
Planorbis corneus 617
Pleiochromia 425
Pleuritis 715
Pleuritis acuta 718
Pleuritis adhesiva 719
Pleuritis chronica 719, 725
Pleuritis fibrinosa 718
Pleuritis granulosa 719
Pleuritis hemorrhagica 718
Pleuritis purulenta 718
Pleuritis serosa-fibrinosa 718
Pleuritis, sicca 718
Pleurocercoid 339
Pleurocercoides Railliet 520
Pleurodynia 727
Pleuro-pericardial friction sounds 723
Pleuro-peritonitis of birds 715
Pleuro-pneumonia, contag., Vol. I ....
Pleuro-pneumonia, contag., septica vitulorum, Vol. I......... Vol. II ....
Pneumatosis bovum 642
Pneumobacillus septicus 685
Pneumoconiosis 707
Pneumoenteritis septica 684
Pneumonia, abortive 658
Pneumonia, catarrhal 671
Pneumonia, croupous 643
Pneumonia, embolic 699
Pneumonia, fibrinous 643
Pneumonia, gangrenous 660, 701
Pneumonia, hypostatic 701
Pneumonia, indurative 707
Pneumonia, insular 671
Pneumonia, interstitial, chronic. 701

	PAGE
Pneumonia, lobar	643
Pneumanio, lobular	671
Pneumonia, mycotic	695
Pneumonia, progredient	659
Pneumonia, suppurative	699
Pneumonia of young animals, septic	684
Pneumonomycosis	695
Pneumonomycosis aspergillina	695
Pneumopericardium	756
Pneumothorax	661, 733
Pneumotborax, closed	660, 735
Pneumothorax, open	660, 735
Pneumothorax, valvular	735
Poikilocytosis	339
Poisoning	197
Poisoning, sausage	212
Poisoning by turnips	101
Polyadenoma, bronchial	630
Polydesmus exitiosus	3
Polymorphus boschadis	403
Polyneuritis, infectious, Vol. III	
Polypi in the intestines	250
Polypi in the nasal cavities	545
Polypi in the stomach	158
Polyvalent immune sera	694
Portal vein, thrombosis of	834
Porthesia chrysorrha	19
Proglottids	343
Protozoan diseases, Vol. III	
Protozoan hepatitis in pigeons	496
Pseudalis capillaris	599
Pseudalis ovis pulmonalis	599
Pseudo foot and mouth disease	14
Pseudo leukemia, Vol. III	
Pseudopericarditis	754
Pseudo tuberculosis, Vol. I	
Pseudo typhus	197
Psittacosis, Vol. III	
Psorospermian rhinitis	555
Ptomaines	339
Ptyalism	27
Puccinia arundinacea	2
Puccinia graminis	2
Puncture of pericardium	753
Puncture of the liver	448
Puncture of the peritoneal cavity	270, 507
Puncture of the pleural cavity	731
Puncture of the rumen	91
Putresin	211
Pyelitis, Vol. III	
Pylorus, stenosis of	150
Pyobacillosis, Vol. I	
Pyopneumothorax	735
Pyosepticemia of sucklings, Vol. I.	
Pyothorax	718
Rabbit infection	536

	PAGE
Ranula inflammatoria	30
Rectal bleeding	170
Rectal catarrh	176
Rectal exploration	313
Rectal inflammation, hemorrhagic	170
Rectal infusion	183
Rectal parasites	315
Regurgitation	36
Reticulitis	123
Reticulitis traumatica	111
Rhabdonema longum	401
Rhabdonema suis	401
Rhachitis, Vol. III	
Rhinitis, catarrhal	523
Rhinitis, chronic, purulent, with atrophy of the turbinated bones in young pigs	536
Rhinitis, coccidial, of rabbits	555
Rhinitis, contagious, of rabbits	536
Rhinitis, croupous	540
Rhinitis, follicular	541
Rhinitis, gangrenous, Vol. III	
Rhinitis, infectious, of swine	532
Rhinitis, phlyctenular	525
Rhinitis, proliferative	525
Rhinitis, purulent	536
Rhinitis, pustular	541
Rhinorrhagia	521
Rhinorrhea	525
Rib furrows	633
Roundworms	356
Rumen, dilatation of	80
Rumen, foreign bodies in	108
Rumen, gas in	86
Rumen, irrigation of	105
Rumen, massage of	84
Rumen, obstruction of	80
Rumen, paralysis of	95
Rumen, puncture of	91
Rumen, section of	86
Ruptura cordis	793
Ruptura vasorum	834
Rupture of liver	436
Rupture of rumen	88
Rupture of stomach	142
Saburra	111, 230
Saccharomyces albicans	17
Saccharomycosis, Vol. III	
Salivation	27
Salt sickness	381
Sand disease	230
Sand in the gall-ducts	431
Sand in the intestines	111, 230
Sand in the stomach	152
Sarcoma of the heart	794
Sarcoma of the esophagus	68
Sarcoma of the intestines	250
Sarcoma of the liver	462

	PAGE
Sarcoma of the lungs	713
Sarcoma of the nasal cavities	545
Sarcoma of the peritoneum	519
Sarcoma of the pharyngeal cavity	45
Sarcosporidiosis	70
Sarcosporidia in heart muscle	795
Sausage poisoning	212
Scalma	569
Schisogony	405
Schweinsberg disease	451
Sclerosis pulmonum	707
Sclerostomiasis, enzootic	258
Sclerostomiasis of horses ...256,	386
Sclerostomum bidentatum	257
Sclerostomum edentatum....256,	386
Sclerostomum equinum	256
Sclerostomum hypostomum	401
Sclerostomum quadridentatum	257
Sclerostomum tetracanthum	386
Sclerostomum vulgare 256,	386
Scolex	343
Sedimentum lateritum	129
Senecio plant	451
Sepsis intestinalis	197
Septicemia of birds, Vol. I	
Septicemia hemorrhagica, Vol. I	
Septicemia of sucklings, Vol. I.	
Sequel of foot and mouth disease	791
Sheep flies	548
Sheep pox, Vol. I	
Shipping fever of horses	621
Sialoadenitis submaxillaris	30
Sialorrhea	27
Simondsia paradoxa	332
Simuliæ	45
Slow heart action	759
Snuffles, infectious, of birds	529
Snuffles, malignant, of rabbits	536
Soor	17
Soor of cattle	17
Soor of sucklings	9
Spasm of the esophagus	50
Spiroptera megastoma	322
Spiroptera mocrostoma	323
Spiroptera pectinifera	336
Spiroptera perforans	336
Spiroptera sanguinolenta 68,	334
Spiroptera scutata	70
Spiroptera strongylina	332
Spiroptera uncinata	337
Splenisation	719
Sporogony	405
Sporotrichum beurmanni	510
Stable pneumonia	671
Staphylococcus pyog. aureus	21
Staphylococci upon walls of blood vessels	822
Stasis hyperemia of the lungs	618
Stenosis ostii aortae	808
Stenosis art. pulm.	810
Stenosis art. ventr. sinistri	810
Stenosis art. ventr. dextri	812
Stenosis ostii venosi dextri	810
Stenosis ostii venosi sinistri	810
Stenosis of the intestines	249
Stenosis of the esophagus	59
Stephanurus dentatus	519
Stilesia centripunctata	345
Stilesia globipunctata	345
Stomacæ	21
Stomach calculi	152
Stomach, catarrh, acute	124
Stomach catarrh, acute, of young animals	183
Stomach catarrh, chronic	132
Stomach catarrh, chronic, of cattle	134
Stomach catherer	145
Stomach catherer, introduction	146
Stomach dilatation, acute, in horses	136
Stomach dilatation, acute, in other animals	147
Stomach dilatation, chronic	149
Stomach diseases	74
Stomach distention 136,	147
Stomach, foreign bodies in	152
Stomach, hemorrhage from	165
Stomach, impaction	217
Stomach, inflammation of	197
Stomach, occlusion of	148
Stomach, parasites of	314
Stomach, rupture of	142
Stomach, sand in	152
Stomach, torsion of	160
Stomach, tumors of	158
Stomach, ulceration of	155
Stomach worms in cattle	330
Stomach worms in deer	330
Stomach worms in goats	323
Stomach worms in sheep	323
Stomach worms in swine	332
Stomatitis, aphthous	9
Stomatitis, aphthous, contagious, Vol. I	
Stomatitis, aphthous, sporadic	6
Stomatitis, catarrhal	1
Stomatitis, erosive 14,	19
Stomatitis, erysipelatous	19
Stomatitis, erythematous	1
Stomatitis, follicular	12
Stomatitis, gangrenous, of lambs	25
Stomatitis, mercurial	23
Stomatitis, mycotic 9,	14
Stomatitis, oidica	17
Stomatitis, papulous, of cattle, infectious	12
Stomatitis, phlegmonous	19

	PAGE
Stomatitis pseudo-aphtosa	14
Stomatitis, pustular, contagious, Vol. I	
Stomatitis, simple	1
Stomatitis, ulcerous 9,	21
Stomatitis, ulcerous, in cattle	14
Stomatitis, vesicular	6
Strangulation of the intestines	272
Strangulation of the spermatic duct	273
Straw eating	214
Streptococcus pyogene	685
Streptococcus serum, Vol. I..... Vol. III	
Streptothricosis, Vol. I	
Streptothrix	686
Stricture of the esophagus	59
Stricture of the intestines	249
Strobila	343
Strongyloides longus	397
Strongyloides westeri	397
Strongylosis, intestinal	397
Strongylosis of lungs	595
Strongylosis of stomach in cattle	330
Strongylosis of stomach in goats	323
Strongylosis of stomach in sheep	323
Strongylus arnfieldi	597
Strongylus axei	323
Strongylus canis bronchialis	599
Strongylus capillaris 599,	600
Strongylus circumcinctus	324
Strongylus commutatus	600
Strongylus contortus 330,	323
Strongylus convolutus	324
Strongylus curticei	324
Strongylus douglasii	336
Strongylus filaria 596,	600
Strongylus follicularis	394
Strongylus longevaginatus	597
Strongylus micrurus 597,	600
Strongylus minutissimus	598
Strongylus neglectus	386
Strongylus nodularis	401
Strongylus oncophorus	330
Strongylus osteragi	324
Strongylus paradoxus 597,	600
Strongylus primitivus	614
Strongylus pulmonalis	597
Strongylus pusillus	599
Strongylus retortæformis	324
Strongylus rubidus	332
Strongylus refescens	600
Strongylus sagittus	597
Strongylus tenuis	401
Strongylus tenuissimus	323
Strongylus tetracanthus	327
Strongylus trachealis	600
Strongylus vasorum	599
Strongylus ventricosus	324
Strongylus vicarius	324
Stuttgart disease of dogs, Vol. I.	
Submaxillary glands, inflammation of	30
Summer dysentery	172
Surra, Vol. I	
Swine erysipelas, Vol. I	
Swine fever, Vol. I	
Swine plague, Vol. I	
Swine pox, Vol. I	
Symplektoptes cysticola	617
Syngamus bronchialis	614
Syngamus laryngeus	614
Syngamus trachealis	614
Synthetocaulus abstrusus	599
Synthetocaulus capillaris	599
Synthetocaulus commutatus	598
Synthetocaulus rufescens	598
Synthetocaulus unciphaurus	599
Syphilis, Vol. I	
Tabanus, Vol. I	
Tabes intestinalis	190
Tachycardia	757
Tænia alba	345
Tænia cesticillus	353
Tænia cœnurus	347
Tænia crassicollis	352
Tænia crassula	353
Tænia cucumerina	348
Tænia denticulata	345
Tænia echinobothrida	353
Tænia echinococcus....347, 464,	710
Tænia elliptica	353
Tænia expansa	345
Tænia infundibuliformis	353
Tænia Krabbei	348
Tænia lineata	349
Tænia mamillana	344
Tænia marginata	347
Tænia ovilla	345
Tænia pectinata	352
Tænia perfoliata	344
Tænia plicata	344
Tænia proglottina	353
Tænia serialis	348
Tænia serrata 347,	491
Tænia struthionis	353
Tænia tetragona	353
Takosis, Vol. I.	
Talma's operation	508
Tapeworms	342
Tapeworms in the lungs	710
Teleangictasia capillaris maculosa	462
Tetanus, Vol. I	
Texas fever, Vol. I	
Thoracocentesis	731
Thrombo-embolic colic	256
Thrombosis of the celiac artery	271

PAGE

Thrombosis of the mesenteric arteries 256
Thrombosis of the mesenteric veins 272
Thrombosis of the nasal veins.. 523
Thrombosis of the portal veins.. 256
Thrombosis of the pulmonary arteries 828
Thrombosis of the vena cava.... 834
Thrombosis of the veins....... 833
Thrombosis with embolism of arteries 828
Thrombosis of axillary arteries. 828
Thrombosis of the carotid arteries 828
Thrombosis of the iliac arteries 828
Thrombus canalisatus 829
Thrombus obturans 829
Thrombus parietalis 829
Thrush 17
Torsion of the abomasum 162
Torsion of the cecum 289
Torsion of the colon 288
Torsion of the intestines 286
Torsion of the rectum 296
Torsion of the stomach 160
Trematodes 355
Trephining of the frontal sinuses 550
Trephining of the nasal sinuses 550
Trichocephalus 373
Trichocephalus affinis 373
Trichocephalus crenatus 373
Trichocephalus depressiusculus. 373
Trichomonas columbæ.......... 420
Trichosoma aërophilum.....555, 599
Trichosoma annulatum......... 73
Trichosoma brevicolle.......... 373
Trichosoma collare............. 373
Trichosoma contortum.......... 73
Trichosoma leporis pulmonale... 598
Trichosoma longicolle........... 373
Trichosoma retusum............ 374
Trichosoma strumosum......... 73
Trichosoma tenuissimum........ 373
Trichotrachelidæ 373
Triodontus 387
Triodontus minor............... 387
Triodontus serratus............. 387
Tropidocerca fissispina......... 337
Trypanosomiasis, Vol. I........
Tuberculosis, Vol. I............
Tuberc...r....... 463

PAGE

Tuberculosis of the nasal membranes 545
Tuberculosis of the pharynx..... 45
Tuberculosis of the stomach.... 159
Tumors, of the heart........... 794
Tumors, esophageal............ 68
Tumors, laryngeal.............. 581
Tumors, liver.................... 461
Tumors, nasal................... 545
Tumors, pharyngeal.............. 45
Tumors, pulmonary............. 713
Tumors of the stomach......... 158
Turnips, disease from eating.... 101
Tympanitis, acute, of ruminants 86
Tympanitis, chronic, of ruminants 93
Typhlocoelum obovale.......... 617
Typhus of the dog, Vol. I......

Ulcer of the stomach........... 155
Uncinaria 374
Uncinaria os papillatam......... 385
Uncinaria radiatum............. 382
Uremia, Vol. I.................

Vaginal catarrh, Vol. I.........
Vagus pneumoata............... 798
Valvular diseases............... 802
Variola, Vol. I.................
Venous occlusion 812
Venous pulse................... 807
Vicia tenuifolia................. 28
Villous heart................... 741
Vitia cordis.................... 802
Volvulus et torsion of the intestine 286
Volvulus of the stomach........ 160
Vomiting 74
Vomiting of blood.............. 77
Vomitus cruentis............... 77

Waltzing motion in intestinal torsion 286
Wild und Rinderseuche, Vol. I..
Wind colic 253
Winslow's foramen, intestines in 279
Winton's disease............... 451
Wool eating, Vol. III...........
Worms in the intestinal canal... 388
Worms in the lungs............ 595
Worms in the stomach......... 323

Yeast cells..................... 207
Yeast fungi..................... 207

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